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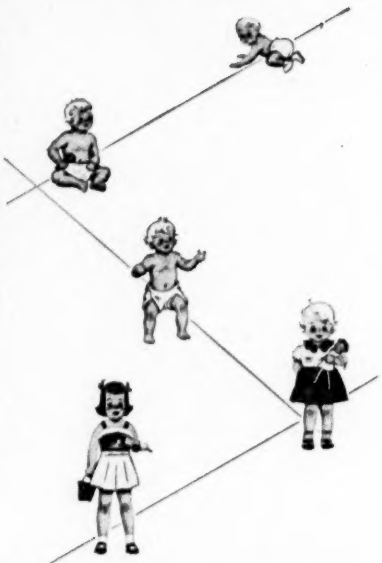

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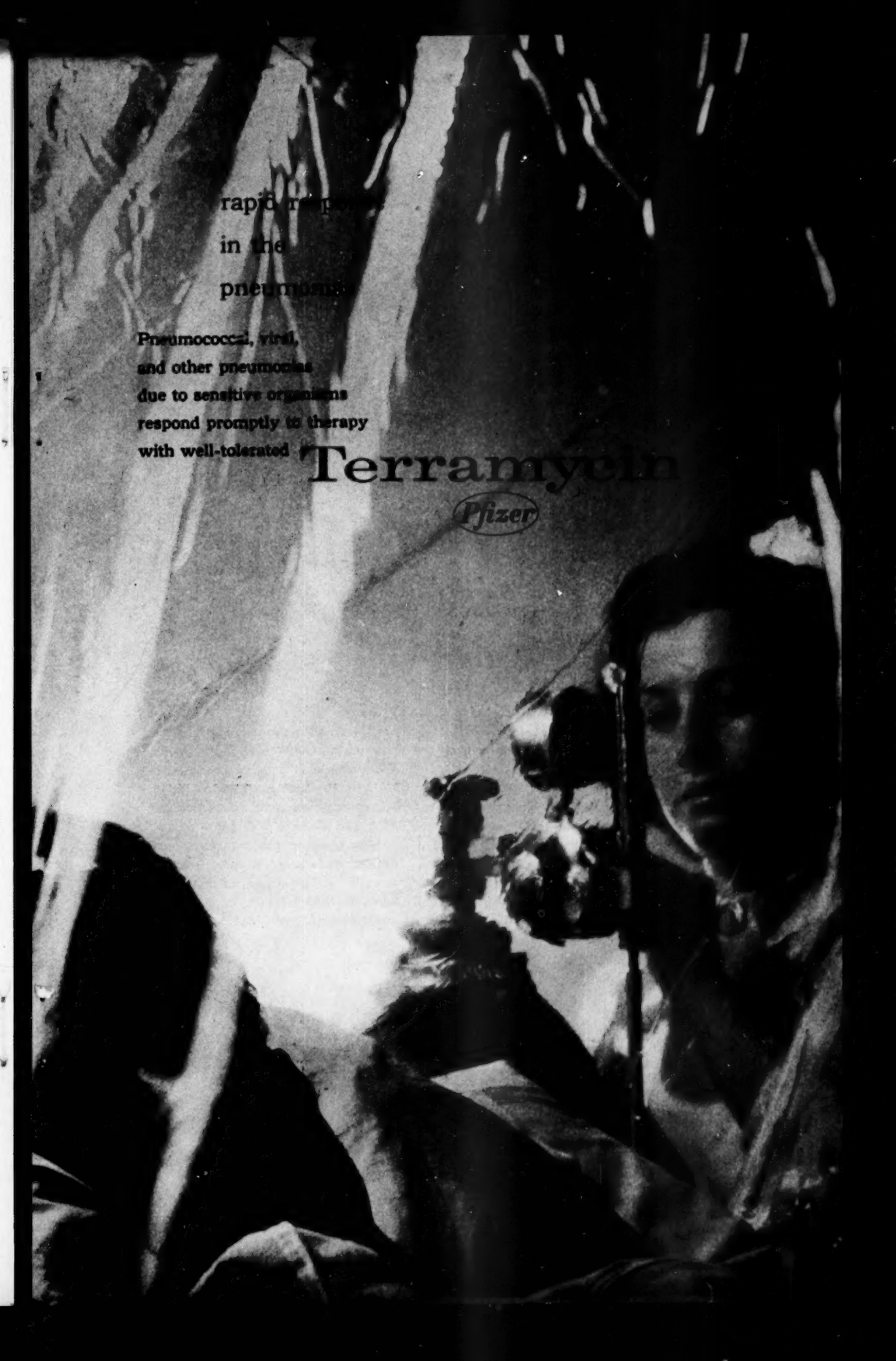
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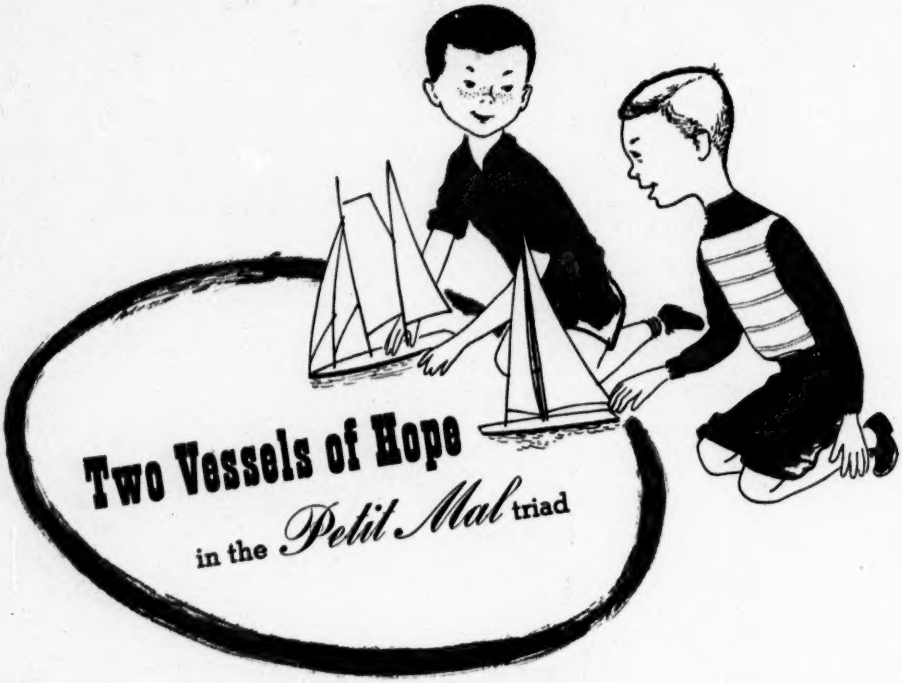

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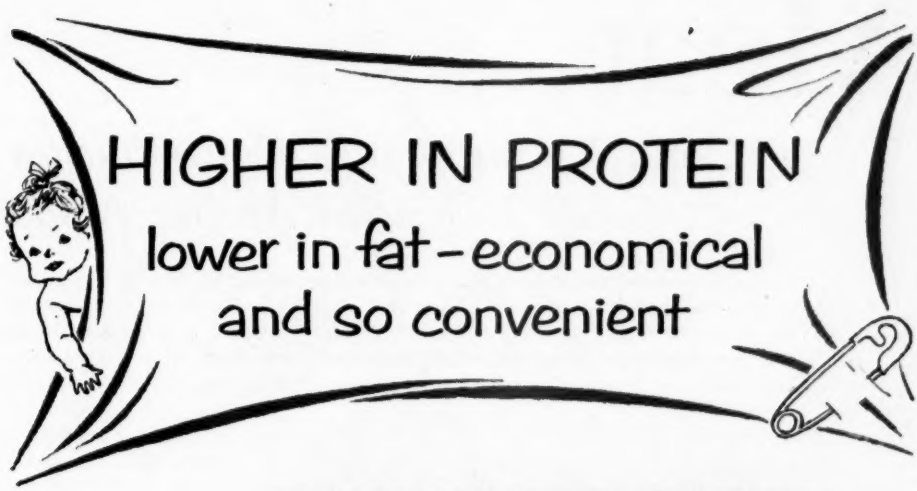

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L-Leucine	150	120	125
L-Lysine	202	163	172
L-Methionine	42	34	40
L-Phenylalanine	70	63	70
L-Threonine	160	169	183
L-Tryptophan	90	90	100
L-Valine	136	107	113
Protein	56	49	51

*In the calculations, averages of the percentages of protein in six different cuts of each type of cooked meat were used, as were averages of the percentages of the amino acids in the protein of the respective cuts.

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2. Rose, W. C.: Half-Century of Amino Acid Investigations, Chem. and Eng. News *30*:2385 (June 9) 1952.
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4. Schweigert, B. S.; Guthneck, B. T.; Kraybill, H. R., and Greenwood, D. A.: The Amino Acid Composition of Pork and Lamb Cuts, J. Biol. Chem. *180*:1077 (Oct.) 1949.

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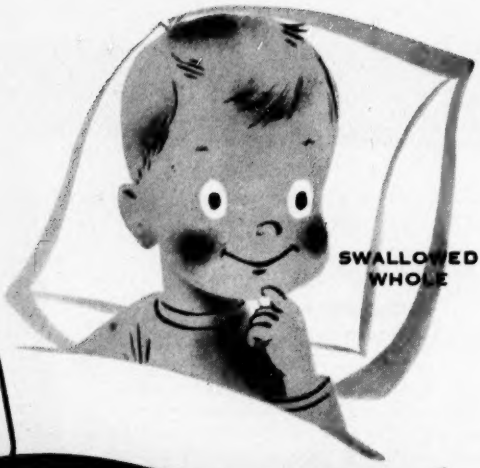

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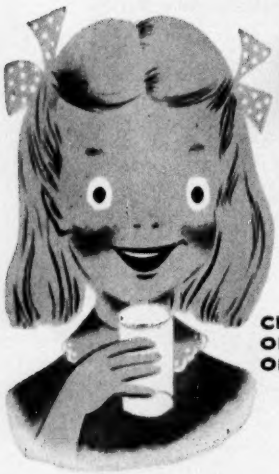

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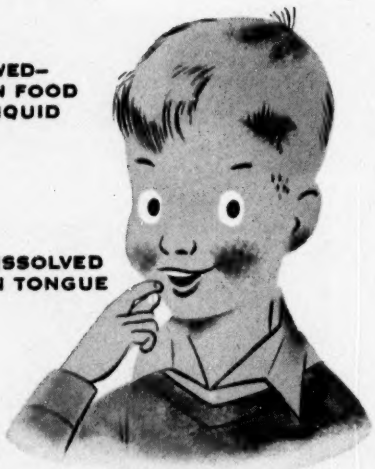

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American Journal of Diseases of Children

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SKELETAL MATURATIONAL PROGRESS OF CHILDREN WITH CHRONIC NUTRITIVE FAILURE

Effect of Dietary Supplement of Reconstituted Milk Solids

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PHILADELPHIA
GEORGE S. PARKER, D.D.S.
AND
CATHERINE CURRIE, B.S.
BIRMINGHAM, ALA.

EVIDENCE that the growth lag of children with chronic nutritive failure may be reduced by the daily addition of milk supplements to inadequate diets has been presented in previous reports from this clinic.[1] In these studies, 41 children with retarded growth associated with chronic undernutrition were given a dietary supplement of reconstituted milk solids equivalent in protein value to that contained in 1 qt. (0.9 liter) of cow's milk. The supplements were given 6 days per week for a period of 20 months. Despite the added milk, the diets of this group remained deficient in calories and in several of the essential nutrients for which standards have been accepted.[1b] Nevertheless, these children gained an average of 1.23 cm. and 1.35 kg. over and above the gains made by ethnically and nutritionally comparable children who did not receive added milk and who served as the reference group.[1b] While improvement in growth progress was widespread and measurable, very few subjects completely reduced their growth lag. Withdrawal of the supplements was followed in almost all instances by a regression in the growth pattern. The submaximal growth responses encountered in the preliminary investigation were thought to be the result of too low a level of supplementation, too short a period of supplementation, dietary imbalance, irreversible changes produced by long-enduring undernutrition, or any combination of these factors.

Associate Professor of Anatomy, Temple University, School of Dentistry (Dr. Snodgrasse).

These studies were supported by grants from the American Dry Milk Institute, Inc. and the Birmingham Citizens Committee.

Northwestern University studies in nutrition at the Jefferson-Hillman Hospital, Birmingham, Ala. From the Department of Nutrition and Metabolism, Northwestern University, Chicago.

1. (*a*) Spies, T. D., and Dreizen, S.: The Effect of Milk Supplements on the Growth of Children with Nutritive Failure: Wetzel Grid Findings, J. Pediat. **34**:393, 1949. (*b*) Dreizen, S.; Currie, C.; Gilley, E. J., and Spies, T. D.: The Effect of Milk Supplements on the Growth of Children with Nutritive Failure: II. Height and Weight Changes, Growth **14**:189, 1950.

The present study was devised to evaluate the influence of these factors on the growth progress of selected young children with chronic nutritive failure and growth retardation. The children selected for this purpose were followed through (*a*) a 40-month period in which they received a dietary supplement equivalent in protein value to 3 qt. (2.8 liters) of milk per week and (*b*) a 6-month period in which an arbitrarily selected number of these children received the equivalent of 12 qt. (11.4 liters) of milk per week while the remainder continued at the previous level. This report is confined to the progress in bone maturation and to the changes in bone texture observed during the 46-month period of supplementation.

MATERIALS AND METHODS

Selection of Patients.—Clinical, dietary, and hematological records, Wetzel grids, and roentgenograms of the hand and wrist of more than 200 children were intensely examined preliminary to selecting the subjects used in this study. Final selection was based on (*a*) clinical, Wetzel Grid or "Red Graph," evidence of growth failure, (*b*) physical evidence of nutritive failure supported by diet records showing severe nutritional inadequacies, (*c*) freedom from debilitating and infectious disease, (*d*) freedom from known food allergies, (*e*) accessibility of the home, and (*f*) cooperation and reliability of the child and his parents.

Forty-one children (19 boys and 22 girls) were selected to receive the dietary supplements. Each was paired with a child who, while subjected to the identical investigative procedure, was not given the added milk. Children of the same sex were paired according to similarity of phase of growth, body build, and social and economic background. The 41 children who received the milk supplements and the 41 controls [2] ranged in age from 1 year to 10 years and 8 months. All were white and of Anglo-Saxon extraction.

Method of Study.—Nineteen children, 9 boys and 10 girls, were given whole milk, and 22 children, 10 boys and 12 girls, received nonfat milk. The supplements were prepared by reconstituting 3 oz. (85 gm.) of nonfat milk solids and 4¼ oz. (120 gm.) of whole milk solids to either one-half pint (240 ml.) or 1 pint volume. The freshly prepared mixtures were refrigerated over night and taken to the homes of the subjects three times per week for a period of 40 months. The total quantity of supplement given each child in the 40-month period was thus equal to the amount provided during the previous study when the equivalent of 6 qt. (5.7 liters) per week was given for 20 months.[1b]

At the beginning of the 41st month of study, and for 6 months thereafter, the weekly amount of milk solids was increased for 19 arbitrarily selected children (6 boys and 13 girls) in the group receiving the supplement to four times that given in the 40 months immediately preceding. (This amount was equal to twice the weekly level of the previous study.) The remaining 22 children continued to receive the dietary supplement equivalent to 3 qt. per week.

To facilitate ingestion of the increased amount of supplement, the method of preparation was altered. One-half the daily amount of milk solids was reconstituted in as small a quantity of water as possible. One-half oz. (14 gm.) of sugar and a few lemon drop chips were added for flavor and palatability. The resultant mixture was frozen to the consistency of ice cream. The remaining half of the daily amount of milk solids were reconstituted to 1 pint or one-half pint volume, and given with the "ice cream" in one supplementary feeding. The supplementary feedings were administered by Catherine Sims, Verna Lee Moore, Martha Hutchinson, Nelwyn Dill, Jane Farley, Merle Babb, and Mabel Morey, each of whom kept an accurate record of the amount of milk ingested. If the supplement was refused in whole or in part at any time for any reason, it was returned to the clinic for disposal. The nutritive value of the supplements is explained in Table 1.

2. The families of four children in the control group, two boys and two girls, moved to other parts of the state before the study was completed, and data pertaining to these children are not included in this report.

The investigative procedure followed in this study was identical with that described in detail in a previous report.[1a] The Todd technique [3] applied to roentgenograms of the hand and wrist was used to establish bone age and to trace skeletal maturation. "Red Graphs" (bone developmental range graphs) [4] depicting the degree of asymmetry of bone development were prepared for each child from roentgenograms taken at four-month intervals during the first 40 study months, and at two-month intervals during the last 6 study months. The Greulich-Pyle Atlas of Skeletal Development of the Hand and Wrist [5] served as the standard of reference. The effect of the various types and concentrations of milk supplements on the rate of bone maturation was determined by comparing the progress made by (*a*) each child given the supplement with that of his control, (*b*) the 19 children whose supplement level was increased during the last 6 months of study with that made by them during the first 40 months, and (*c*) the 19 children receiving the increased amount of supplement with that of the 22 whose level remained unchanged.

Changes in bone texture were determined by examination and comparison of roentgenograms taken of each individual at the beginning of supplementation and at the end of the 40th and 46th study months. The changes were evaluated in terms of the thickness of the cortical bone, amount and texture of the spongy bone, presence of radiolucent areas, degree of calcification, and the presence of transverse radiopaque lines in the distal end of the radius.

TABLE 1.—*Nutrients Supplied by Weekly Dietary Supplements of Whole or Nonfat Dry Milk**

Nutrient	Nonfat Milk		Whole Milk	
	3 Qt./Wk.	12 Qt./Wk.	3 Qt./Wk.	12 Qt./Wk.
Calories	916.80	3,887.20	1,800.90	7,373.60
Protein	94.20 gm.	376.80 gm.	95.40 gm.	381.60 gm.
Calcium	3.30 gm.	13.20 gm.	3.48 gm.	13.92 gm.
Phosphorus	1.92 gm.	7.68 gm.	2.76 gm.	11.04 gm.
Vitamin A	.....	.....	3,941.70 I. U.	15,766.80 I. U.
Thiamin	0.90 mg.	3.60 mg.	0.96 mg.	3.84 mg.
Riboflavin	5.19 mg.	20.76 mg.	5.34 mg.	21.36 mg.
Niacin	2.37 mg.	9.48 mg.	2.46 mg.	9.84 mg.
Vitamin C	.....	.....	35.10 mg.	140.40 mg.

* 170 calories per week supplied by sugar and lemon drop chips added as flavoring agents.

OBSERVATIONS

Changes in Rate and Symmetry of Bone Maturation.—The bone maturity status of the 82 children just prior to the initiation of the dietary supplement is shown in Table 2. Sixteen of the 19 boys and 19 of the 22 girls scheduled to receive the added milk had a chronological age greater than their skeletal age. Skeletal age exceeded chronological age in 3 boys and 2 girls in the control group. The mean skeletal age lag in the boys selected for the whole and nonfat milk supplements was 11.9 and 13.5 months respectively. Corresponding values for the girls were 6.0 months and 11.4 months. The mean skeletal age lag of the boys and girls comprising the control group was 7.8 months and 9.9 months respectively.

Red Graphs from roentgenograms taken at the beginning of supplementation showed that each of the 82 children had a substantial amount of asymmetry in bone

3. Todd, T. W.: Atlas of Skeletal Maturation Hand, St. Louis, C. V. Mosby Company, 1937.

4. Pyle, S. I.; Mann, A. W.; Dreizen, S.; Kelly, H. J.; Macy, I. G., and Spies, T. D.: A Substitute for Skeletal Age (Todd) for Clinical Use: The Red Graph Method, J. Pediat. **32**:125, 1948.

5. Greulich, W. W., and Pyle, S. I.: Radiographic Atlas of Skeletal Development of the Hand and Wrist Based on the Brush Foundation Study of Human Growth and Development Initiated by T. Wingate Todd, Stanford, Stanford University Press, 1950.

development as evidenced by the width of the zone between the upper and lower level lines. The mean values for the Red Graph zones were 29.2 months for boys selected to receive whole milk, 24.1 months for boys selected to receive nonfat milk, and 28.4 months for the control boys. Corresponding values for the girls were 15.9 months, 18.3 months, and 17.3 months.

TABLE 2.—*Status of Skeletal Maturation at the Beginning of the Administration of Whole and Nonfat Milk Supplements*

	Maturational Status, Mo.							
	Chronological Age		Skeletal Age		Difference*		Zone†	
Subjects	Mean	Range	Mean	Range	Mean	Range	Mean	Range
Test boys								
Whole	86.00	14-129	74.11	12-117	−11.89	+ 4 to −25	29.22	6-58
Nonfat	82.70	37-119	69.20	22-120	−13.50	+15 to −26	24.10	15-29
Total	84.26	14-129	71.53	12-120	−12.74	+15 to −26	26.53	6-58
Control boys	81.74	21-118	73.88	19-127	− 7.76	+11 to −29	28.35	18-51
Test girls								
Whole	77.20	13-120	71.20	11-116	− 6.00	+ 4 to −15	15.90	6-30
Nonfat	76.17	29-125	64.75	22-127	−11.42	+ 2 to −30	18.25	9-38
Total	76.64	13-125	67.68	11-127	− 8.98	+ 4 to −30	17.18	6-38
Control girls	79.25	13-127	69.35	19-122	− 9.90	+ 6 to −33	17.25	9-30

* + indicates skeletal age greater than chronological age.
− indicates skeletal age smaller than chronological age.
† Measurements obtained from "Red Graph."

TABLE 3.—*Progress Made in Bone Maturation by Forty-One Children Receiving Milk Supplements and Thirty-Seven Control Children in the First Forty Study Months* *

	Skeletal Maturation, Mo.							
	Skeletal Age		Upper Level†		Lower Level†		Zone‡	
Subjects	Mean	Range	Mean	Range	Mean	Range	Mean	Range
Test boys								
Whole	40.00	32-58	37.89	24-60	45.78	17-84	−7.89	+21 to −43
Nonfat	38.20	26-53	34.90	24-56	35.70	19-69	−0.80	+20 to −30
Total	39.05	26-58	36.32	24-60	40.47	17-84	−4.16	+21 to −43
Control boys	37.65	26-55	35.88	27-51	37.94	14-55	−1.88	+19 to −16
Test girls								
Whole	41.10	26-61	39.50	23-47	40.20	22-60	−0.70	+14 to −18
Nonfat	39.58	28-49	39.00	30-51	39.22	23-51	−0.92	+24 to −14
Total	40.27	26-61	39.23	23-51	40.05	22-60	−0.82	+24 to −18
Control girls	41.05	29-56	40.25	28-51	39.35	15-59	+0.90	+27 to −14

* Nutritional equivalent of supplement level: 3 qt./wk.
† "Red Graph" measurements.
‡ + indicates increase in amount of zone.
− indicates decrease in amount of zone.

The progress in bone maturation made by each group during the first 40 study months is presented in Table 3. Very little difference was noted between the mean increases in skeletal age made by the children receiving the milk supplements and that made by those who comprised the control group. Mean skeletal age gains made during this period were 40.0 months for boys receiving whole milk, 38.2 months for boys receiving nonfat milk, and 37.7 months for the control boys. Corresponding values for girls were 41.1 months, 39.6 months, and 41.0 months.

Comparison of the Red Graph progress made by each group during the 40-month period showed essentially no difference between the mean increases in upper and lower level and in the mean extent of the zone between the children given the nonfat milk supplement, the girls given the whole milk supplement, and those not provided with added milk. The mean increase in lower level for the boys given whole milk exceeded that of the control group by 7.8 months. The mean decrease in zone in this group exceeded that of the control group during the same period by 6.0 months. The significance of these differences is questionable since the mean values were greatly influenced by one child in whom an absent bone center appeared, and who subsequently made a gain in lower level of 84 skeletal months in 40 calendar months.

Although there was a close correspondence in the mean values of each group for the increases in skeletal age and changes in Red Graph during the first 40 months, a great variation in individual response was encountered during this phase. Thus, while 11 boys and 9 girls who received the milk supplements made greater gains in skeletal age than did their paired controls, the gains made by 6 boys and 9 girls in the control group exceeded that of the children provided with the milk supplements. A similar lack of correlation was noted in the Red Graph measurements for this period.

The effect of increasing the supplement level on the rate of bone maturation and on the symmetry of skeletal development is shown in Table 4. The mean increase in skeletal age made by the boys given the increased amount of whole milk (protein equivalent of 12 qt. per week) exceeded that of the boys whose supplement level remained unchanged (protein equivalent of 3 qt. per week) by 4.9 months and that of their paired controls by 5.5 months during the six-month period. Similarly, the mean skeletal age increase of the girls given the protein equivalent of 12 qt. of whole milk per week exceeded that of the girls given the lesser amount by 6.8 months, and that of the control groups by 5.4 months. At the 12-qt. level of nonfat milk supplementation, the boys underwent a mean skeletal age increase of 9.3 months as compared with 5.0 months for their paired controls and 6.2 months for those whose supplement level remained unchanged. Corresponding mean values for the girls were 10.5 months, 5.7 months, and 6.3 months respectively. It is noteworthy that each of the 19 children, 6 boys and 13 girls, whose supplement level was increased made moderately to substantially greater gains in skeletal age than did their paired controls during the last six study months.

During the same period differentials similar to those noted for skeletal age were encountered in the mean increases in the upper and lower levels of the Red Graph in the various groups of children. The mean increments in the maturation of the most advanced and least advanced bone centers of the children given the increased amount of either whole milk solids or nonfat milk solids were substantially greater than those made by the group continued at the original supplement level and by the control groups. There was no significant difference between the mean values for these measurements in the girls receiving whole milk and the values in those receiving nonfat milk. The smallness of the sampling rendered a similar comparison for the boys impractical. For the most part, the gains in the upper level in these children exceeded those in the lower level, with a resultant increase in the width of the zone.

As a group, the boys receiving the whole milk increased their mean monthly rate of bone maturation from 1.00 bone month per calendar month during the first

40 study months to 1.75 bone months per calendar month during the last 6 study months. Corresponding changes for the boys receiving nonfat milk were 0.85 to 1.54 bone months per calendar month, and for the boys serving as controls, 0.94 to 0.86 bone months per calendar month. Corresponding values for the girls were 1.00 to 1.86, 0.99 to 1.75, and 1.03 to 0.94 bone months per calendar month respectively. The mean changes in the upper and lower levels on the Red Graph for each group were similar in direction and magnitude to those observed for skeletal age (Tables 3 and 4).

TABLE 4.—*Progress Made in Bone Maturation by Forty-One Children Receiving Milk Supplements and Thirty-Seven Control Children During the Last Six Study Months* *

	Skeletal Maturation, Mo.							
	Skeletal Age		Upper Level†		Lower Level†		Zone‡	
Subjects	Mean	Range	Mean	Range	Mean	Range	Mean	Range
Supplement Level, 3 Qt./Wk.								
Test boys								
Whole	5.57	4-10	6.00	4- 9	2.57	0-6	+3.43	+6 to 0
Nonfat	6.17	5- 9	5.67	3-12	5.17	2-8	+0.50	+9 to −5
Total	5.85	4-10	5.85	3-12	3.77	0-8	+2.08	+9 to −5
Control boys	5.27	4- 6	5.09	0- 8	3.45	0-6	+1.64	+6 to −6
Test girls								
Whole	4.33	2- 8	6.33	3-12	3.33	0-6	+3.00	+6 to 0
Nonfat	6.33	3- 9	5.17	2- 9	4.00	3-6	+1.17	+6 to −3
Total	5.67	2- 9	5.56	2-12	3.78	0-6	+1.78	+6 to −3
Control girls	5.57	4- 8	5.14	2- 7	5.43	2-9	−0.29	+4 to −4
Supplement Level, 12 Qt./Wk.								
Test boys								
Whole	10.50	9-12	8.50	8- 9	12.50	9-16	−4.00	0 to −8
Nonfat	9.25	6-15	10.25	7-15	9.00	5-12	+1.25	+4 to −1
Total	9.67	6-15	9.67	7-15	10.17	5-16	−0.50	+4 to −8
Control boys	5.00	3- 6	5.67	3- 8	3.83	0- 6	+1.83	+8 to −2
Test girls								
Whole	11.14	9-14	10.29	6-12	7.43	3-15	+2.86	+9 to −6
Nonfat	10.50	8-15	11.33	6-16	7.00	3-15	+4.33	+10 to −6
Total	10.85	8-15	10.77	6-16	7.23	3-15	+3.54	+10 to −6
Control girls	5.69	2- 9	5.46	2-12	5.46	2-9	0.00	+6 to −3

* 22 children received nutritional equivalent of 3 qt./wk.
19 children received nutritional equivalent of 12 qt./wk.
† "Red Graph" measurements.
‡ + indicates increase in amount of zone.
— indicates decrease in amount of zone.

The changes in the rate of bone maturation in each child are shown in Chart 1. Every child who received the increased amount of supplement showed a substantial increase in the rate of bone maturation during the last 6 study months, regardless of sex or type of milk offered. In contrast, 11 of the 19 control children paired with those given the supplement level of 12 qt. per week underwent a deceleration in the rate of bone maturation during this period. The rate for one child remained unchanged, and in the remaining seven the increase ranged from slight to moderate.

Seventeen of the 19 children (89%) given the increased quantity of supplement underwent a notable increase in the rate of maturation of their most advanced bone center during the last six study months. In contrast, 10 (45%) of the children

maintained at the supplement level equivalent to 3 qt. per week and 17 (46%) of the paired controls underwent a similar notable change during the same period (Chart 2).

Acceleration in the rate of maturation of the least advanced bone center was shown by 11 (58%) of the children at the 12-qt. level of supplementation, 5 (23%) at the 3-qt. level of supplementation, and 11 (30%) of the control children (Chart 3).

Five children (26%) at the 12-qt. level of supplementation, 6 (27%) at the 3-qt. level of supplementation, and 10 (27%) control children decreased the width of their Red Graph zone during the last six study months.

Changes in Bone Texture.—Definite deficiencies in bone quality were seen in the roentgenograms taken at the beginning of the administration of the milk supple-

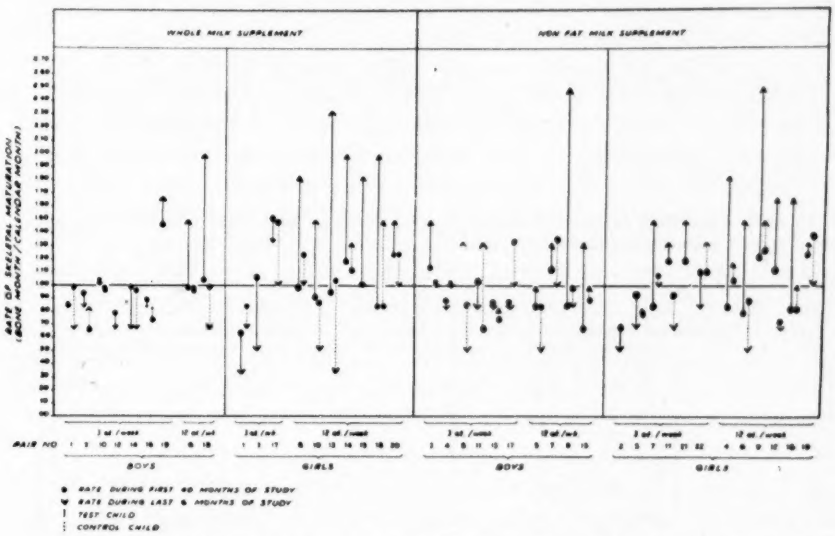

Chart 1.—Individual changes in rate of skeletal maturation during the two phases of milk supplementation.

ments. The thickness of the cortical bone of the metacarpal shafts was submedium in 31% of the subjects selected for supplementation and in 54% of the control group. In no child could the quality of the cortical bone be considered good. The spongy bone was subnormal in quantity in 54% of the group scheduled to receive the supplements and in 70% of the control group. In both groups the spongy bone frequently had a "cindery" appearance. This was particularly obvious in the carpals and to a lesser extent in the metacarpals and distal ends of the radius and ulna. Radiolucent areas, indicative of a deficiency in calcium deposition, were observed in 66% of the children chosen for supplementation and in 81% of the control group. The more obvious areas of radiolucency were small; the less obvious were somewhat larger, thereby producing a cloudy overall appearance. Calcification was submedium to poor in 66% of the children selected for supplementation and in 70% of the control group. The distal ends of the radius of 16 children chosen to receive

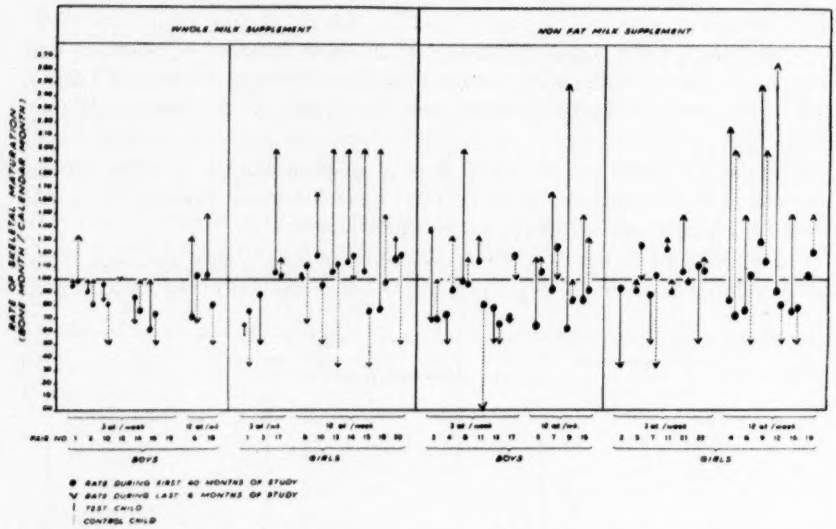

Chart 2.—Individual changes in rate of increase in upper level (Red Graph [4]) during the two phases of milk supplementation.

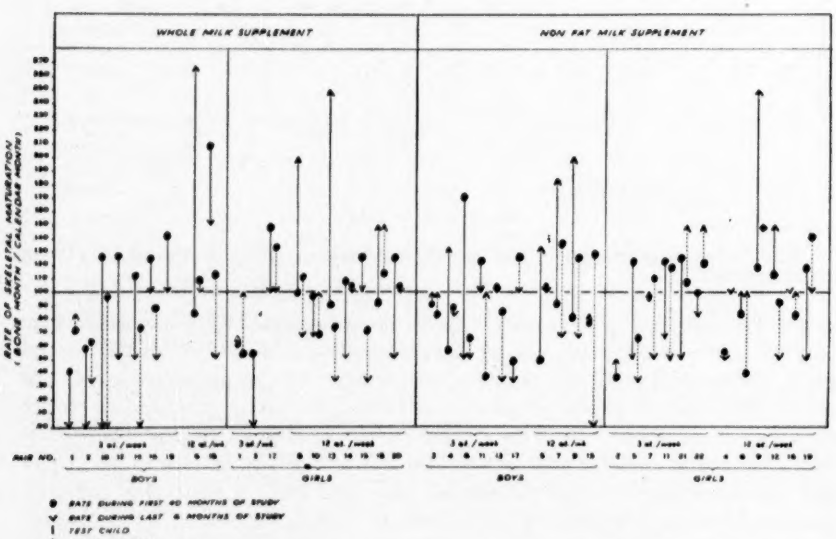

Chart 3.—Individual changes in rate of increase in lower level (Red Graph [4]) during the two phases of milk supplementation.

supplements and 9 of the control children contained one or more transverse lines of increased density which, in the presence of corroborative evidence, is usually regarded as indicative of previous arrest or pronounced retardation of bone growth. Although such lines are frequently associated with undernutrition, none of the lines observed in either group was particularly heavy, and rarely did they extend across the entire width of the shaft.

No notable uniform improvement in bone texture and quality was observed during the periods of supplementation. The quality of the cortical bone remained essentially unchanged throughout the 46-month study period regardless of the type and amount of supplement supplied. No change was visible in the spongy bone during the first 40 study months, and only one child, receiving the supplement level of 12 qt. per week, showed improvement in the amount and texture of the spongiosa. No noteworthy decrease in the number and extent of radiolucent areas was observed during the first 40 study months either in the children receiving the added milk or in the children with whom they were paired. During the last six study months, however, the radiolucent areas disappeared in 35% of the children given the increased amount of supplement, in 25% of the children maintained at the 3-qt. level, and in 10% of the control children. Calcification as manifested in the metacarpal shafts remained essentially unchanged until the period of increased supplementation, when a slight improvement was noted in 35% of the children receiving this supplement. The transverse lines of arrested growth decreased in number in 10 children and increased in number in 6 receiving the added milk during the first 40 study months. In the same period, seven children in the control group developed additional transverse lines in the distal end of the radius and only one child showed a decrease. In the last six study months, a decrease was noted in all children showing such lines in the group given the increased amount of supplement in contrast to the appearance of new lines in two children maintained at the 3-qt. level, and in six children in the control group.

COMMENT

The present findings clearly demonstrate that alteration in the pattern of skeletal maturation in children with nutritive failure may be reversed to a considerable degree by the supplementary feeding of reconstituted whole or nonfat milk solids. The removal of deficiencies of skeletal maturation in these children, regardless of age, appears to be primarily dependent upon the adequacy of the level of supplementation. Thus, children with chronic nutritive failure given supplements of cow's milk at a level equivalent in protein value to 3 qt. per week for a period of 40 months, showed little if any change in rate of bone maturation as contrasted with that of a comparable group not receiving the added milk. A fourfold increase in the quantity of milk supplements for a period of six months resulted in a readily detectable improvement in the overall rate of bone maturation in each of 19 children given the increased quantity of supplement.

Improvement in rate of skeletal maturation occurred independent of a similar change in the symmetry of bone development as measured by the Red Graph. The frequency of decrease in the width of the Red Graph zone in the children given the protein equivalent of 12 qt. of milk per week was similar to that noted in the control group and in the group given one-fourth this amount of supplement. In the majority

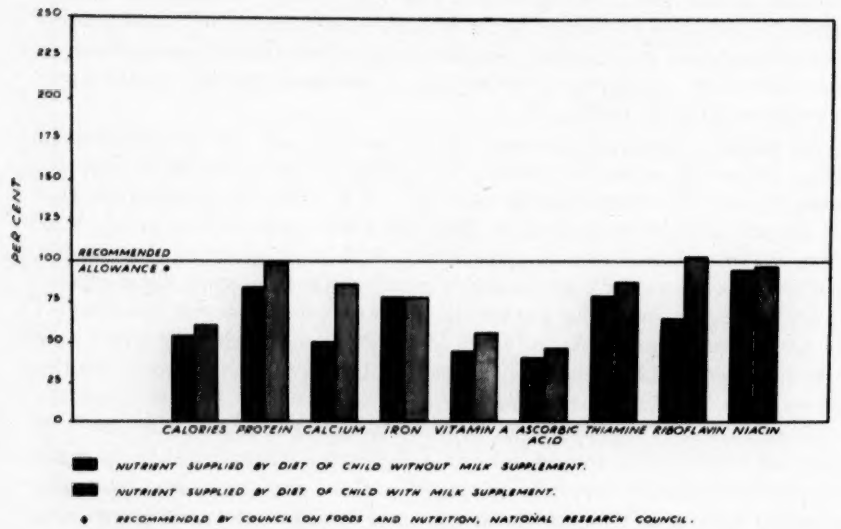

Chart 4.—Nutrients supplied by daily diet of child with and without whole milk supplement equivalent to 3 qt. of milk per week.

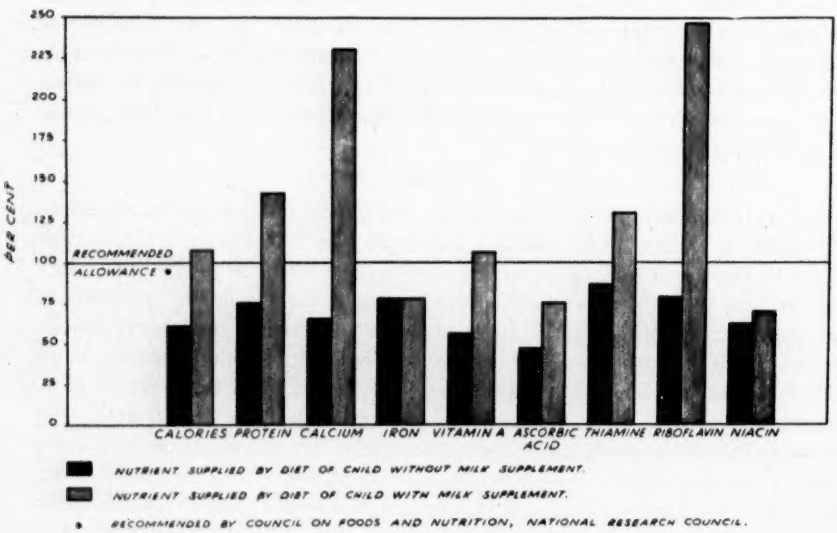

Chart 5.—Nutrients supplied by daily diet of child with and without whole milk supplement equivalent to 12 qt. of milk per week.

of instances the most advanced bone centers were accelerated to a greater degree than the least advanced bone centers, resulting in an increase in the width of the zone. The addition of the nutrients contained in the milk solids was not accompanied, therefore, by a preferential acceleration of the most retarded centers.

The mean increases in skeletal age during the last 6 study months made by the children given whole milk and those given nonfat milk supplements were similar in magnitude despite a daily differential of approximately 600 Cal. between the two types of milk. Each supplement contained similar amounts of calcium, phosphorus, and protein (Table 1). These levels together with those derived from the daily home diets were sufficient to approach or exceed the recommended allowances for these nutrients based on chronological age [6] (Charts 4 and 5). The uniform spurt in the rate of skeletal maturation noted in the children receiving the equivalent of 12 qt. of whole or nonfat milk appears to be an expression of the increase in availability of the food factors concerned with bone formation rather than one of mere caloric intake.

In addition to the retardation prevailing in skeletal age prior to the administration of the supplements, all the hand skeletons appeared to be inadequately mineralized. While little if any change occurred during the first 40 study months in the children receiving the supplements, slight improvement in bone texture was noted in some of the children during the period when the 12-qt. level of supplement was supplied. It would thus appear that the available mineral was utilized for bone maturation rather than for an increase in bone density. This premise is qualified, however, by the observation that fluctuations in some aspects of bone texture are of slow production, requiring a year or more for the development of plainly discernible differences.[3]

SUMMARY AND CONCLUSIONS

Nineteen children with nutritive failure and retarded growth increased their mean rate of skeletal maturation by 80% (boys, 79%; girls, 81%) during a six-month period in which they received a dietary suppliment of dry milk solids equivalent in protein value to 12 qt. of milk per week. In the 40 months immediately preceding, during which their progress in skeletal maturation was not remarkable, each child had received the same type of supplement at a level of 3 qt. per week.

During the six-month period the mean increase for the boys receiving the increased supplement exceeded that of their paired controls by 93% and that of boys continuing to receive the 3-qt. level of supplementation by 65%. The mean increase for the girls exceeded that of their paired controls by 90% and that of girls receiving the lesser supplement level by 90%.

Seventeen of the 19 children given the 12-qt. level of supplementation increased the rate of maturation of their most advanced bone centers, thereby reducing the lag between their actual and genetically-limited growth rate. The increases in upper level were greater both in frequency and in magnitude than those in the lower level, indicating a lack of preferential utilization of nutrients by the most retarded bones in the skeletal area.

6. The dietary analyses were made by Miss Cleo Arnett and were based on her personal observations of the quality, quantity, and method of preparation of the food used by each family.

The children receiving reconstituted nonfat milk made essentially the same amount of progress in bone maturation as those given reconstituted whole milk.

Despite differences in nutritional requirements at different ages, no relationship was noted between chronological age and the degree of increase in bone maturation.

Children receiving the dietary supplements in quantities equivalent to 3 qt. of milk per week for a period of 40 months made no greater improvement in rate of bone maturity or in bone texture than did a control group not given the added milk.

Only scattered slight improvement in bone texture was observed in the group receiving the 12qt. level of supplementation. It would appear that the bone-forming nutrients contained in the supplement were devoted to an increase in bone maturation rather than to an increase in bone density.

The retarding effect of prolonged nutritive failure on skeletal maturation is to a great extent reversible provided the nutrient value of the dietary supplement is sufficient to overcome the accumulated deficiencies.

PROLONGED OBSTRUCTIVE JAUNDICE IN INFANCY

III. Liver Function Tests

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AND
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IN A PREVIOUS report,[1] we have reviewed the cases of 156 infants admitted to the Children's Medical Center because of prolonged obstructive jaundice. This series consisted of 94 cases of biliary atresia, 23 cases of inspissated bile due to erythroblastosis, 30 cases of inspissated bile of unknown cause, 4 cases of infectious hepatitis, and 5 cases of obstructive jaundice due to miscellaneous causes. The present study is an analysis of the liver function tests performed on these infants and an attempt to demonstrate the usefulness and limitations of these tests in the differential diagnosis of obstructive jaundice in infancy.

METHODS AND MATERIALS

The criteria for the selection of cases have been presented previously. The series as a whole included all of the cases of infants who were thought to have prolonged jaundice due to some type of obstruction; most of the infants were admitted to the hospital with a tentative diagnosis of biliary atresia.

The liver function tests performed on these infants are listed in Table 1.[2] All of the tests requiring serum can be done on 1.0 cc. of blood.

From the Medical Service of the Children's Medical Center, the Department of Pediatrics of the Beth Israel Hospital, and the Department of Pediatrics of the Harvard Medical School. Aided by a grant from Mead Johnson & Company.

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2. (*a*) Harrison, G. A.: Chemical Methods in Clinical Medicine: Their Application and Interpretation with the Technique of Simple Tests, Ed. 2, London, J. & A. Churchill, Ltd., 1937. (*b*) Godfried, E. G.: Clinical Tests for Bilirubin in Urine, Biochem. J. **28**:2056, 1934. (*c*) Wallace, G. B., and Diamond, J. S.: Significance of Urobilogen in Urine as Test for Liver Function, with Description of Simple Quantitative Method for Its Estimation, Arch. Int. Med. **35**:698 (June) 1925. (*d*) Bodansky, A.: Phosphatase Studies: Determination of Serum Phosphatase; Factors Influencing Accuracy of Determination, J. Biol. Chem. **101**:93 (June) 1933. (*e*) Maclagan, N. F.: Thymol Turbidity Test: New Indicator of Liver Dysfunction, Nature, London **154**:670 (Nov. 25) 1944. (*f*) Neefe, J. R.: Results of Hepatic Tests in Chronic Hepatitis Without Jaundice: Correlation with Clinical Course and Liver Biopsy Findings, Gastroenterology **7**:1 (July) 1946. (*g*) Hanger, F. M.: Serological Differentiation of Obstructive from Hepatogenous Jaundice by Flocculation of Cephalin-Cholesterol Emulsions, J. Clin. Invest. **18**:261 (May) 1939. (*h*) Malloy, H. T., and Evelyn, K. A.: Determination of Bilirubin with Photoelectric Colorimeter, J. Biol. Chem. **119**:481 (July) 1937. (*i*) Ducci, H., and Watson, C. J.: Quantitative Determination of Serum Bilirubin with Special Reference to Prompt-Reacting and Chloroform-Soluble Types, J. Lab. & Clin. Med. **30**:293 (April) 1945.

RESULTS

I. *Bile and Urobilinogen Tests.*—The results of the tests on urine bile, urobilinogen, and stool bile are shown in Table 2.

Urine Bile: All of the infants were found to have bile present in the urine except one infant with thrombosis of the portal vein who was only mildly jaundiced.

Urine Urobilinogen: Eight of the 26 infants with biliary atresia, 3 of 4 infants with inspissated bile due to erythroblastosis, and 5 of 6 infants with inspissated bile of unknown cause had urobilinogen in the urine. This was present as a trace in undiluted urine to positive in dilutions up to 1:80, with most of the infants in the range of less than 1:4.

TABLE 1.—*Liver Function Tests*

Group	Material	Test	Method	Normal Range
I	Urine	Bile	Harrison,[2a] Godfried [2b]	Negative
	Urine	Urobilinogen	Wallace and Diamond [2c]	Trace to 1:40 dilution
	Stool	Bile	Harrison,[2a] Godfried [2b]	Positive
II	Serum	Alkaline phosphatase	Bodansky [2a]	5 to 12 Bodansky units
III	Serum	Thymol turbidity	Maclagan [2e]	0 to 4 units at the end of 30 min.
		Thymol flocculation	Neefe [2f]	0 to 1+ at the end of 18 hr.
		Cephalin-cholesterol flocculation	Hanger [2g]	0 to 2+ at the end of 24 hr.
IV	Serum	One-minute bilirubin	Malloy and Evelyn [2h] as modified by	0 to 0.2 mg./100 cc.
		Total serum bilirubin	Ducci and Watson [2i]	0 to 0.8 mg./100 cc.

TABLE 2.—*Bile and Urobilinogen Studies*

Diagnosis	Urine Bile			Urine Urobilinogen			Stool Bile			
	Total	Positive	Negative	Total	Positive	Negative	Total	Positive	Doubt	Negative
Biliary atresia	60	60	0	26	8	18	66	0	0	66
Inspissated bile due to erythroblastosis fetalis	16	16	0	4	3	1	11	3	3	5
Inspissated bile of unknown cause	26	26	0	6	5	1	26	1	2	23
Miscellaneous	6	5	1	3	1	2	5	1	0	4
Total	108	107	1	39	17	22	108	5	5	98

Stool Bile: Bile in the stool was classified as positive, doubtful, or negative. The doubtful cases included those in which bile appeared either infrequently or in such small amounts as to be questionable. Of the 66 infants with atresia of the bile ducts tested, none passed bile into the stool. Of the 11 infants with inspissated bile due to erythroblastosis, 3 had definite and 3 others had doubtful tests for bile in the stools. Of the 26 infants with inspissated bile of unknown cause, 1 had definite and 2 others had doubtful tests for bile in the stool. In the miscellaneous group, only the patient with thrombosis of the portal vein had bile in the stool. It is interesting to note that in all cases of biliary atresia operated on successfully, bile appeared in the stools in normal amounts within 2 weeks after surgery.

II. *Alkaline Phosphatase.*—The alkaline phosphatase values obtained are listed in Table 3. Although the values were in general higher than in normal infants, there was essentially no difference in the individual or average figures for the four groups of cases. Serial determinations in individual patients were also highly variable and did not reflect a trend. A number of patients with biliary atresia showed values which were within the normal range for infants.

III. *Flocculation Tests.*—The results of the flocculation tests are listed in Table 4. The tests were taken as a whole and interpreted as positive, doubtful, or negative. The tests were regarded as positive when two or three of the flocculation tests were strongly positive, doubtful when only one of the flocculation tests was positive, and negative when all of the flocculation tests were within normal limits.

Among the 35 infants with biliary atresia tested on admission, there were no positive tests and only one doubtful one. It is noteworthy that all five infants who were found to be inoperable and developed cirrhosis subsequently showed positive flocculation tests. Of these, one became positive at 6 months and others at one year of age. In the patients with inspissated bile due to erythroblastosis, there were no positive and three doubtful tests among 21 infants. In the 15 infants with inspissated bile of unknown cause, there were no positive and two doubtful tests. All three infants in this group in whom cirrhosis developed subsequently showed positive

TABLE 3.—*Alkaline Phosphatase Determinations*

Diagnosis	No. of Cases	Bodansky Units		
		Mean	Maximum	Minimum
Biliary atresia	25	25.9	100.1	5.5
Inspissated bile due to erythroblastosis fetalis	4	16.0	24.5	10.6
Inspissated bile of unknown cause	11	28.1	85.2	12.2
Miscellaneous	1	47.0	.....	...
Total	41	26.0	100.1	5.5

TABLE 4.—*Liver Function Flocculation Tests*

Diagnosis	No. of Cases	Positive	Doubtful	Negative
Biliary atresia (early)	35	0	1	34
Biliary atresia with cirrhosis	5	5	0	0
Inspissated bile due to erythroblastosis fetalis	21	0	3	18
Inspissated bile of unknown cause	15	0	2	13
Inspissated bile with cirrhosis	3	3	0	0
Miscellaneous (hepatitis)	3	1	1	1
Other causes	4	0	0	4
Total	86	9	7	70

flocculation tests. Of the three infants with infectious hepatitis, one showed positive, one showed doubtful, and one showed negative tests. All five infants with miscellaneous causes of obstructive jaundice showed negative flocculation tests.

IV. *Serum Bilirubin Levels.*—The serum bilirubin levels prior to 1947 were determined by the method of Malloy and Evelyn [2b] and subsequently by the method of Ducci and Watson.[21] Since the direct serum bilirubin levels obtained by the first method are higher than those obtained by the second method, while the total serum bilirubin levels obtained by both methods are identical, it was decided to analyze the latter data only. All of the infants studied had approximately equal elevation of both the direct and the indirect bilirubin fractions, which is characteristic of obstructive jaundice.

Table 5 illustrates the average bilirubin levels obtained in infants of various ages with obstructive jaundice. The infants with biliary atresia showed low initial bilirubin levels, which rose slowly but steadily over many months. The infants with

TABLE 5.—*Average Total Serum Bilirubin Levels*

Diagnosis	Age, Mo.															
	1		2		3		4		5		6 to 8		9 to 12		13+	
	Mg./ 100 Cc.	No. of Cases	Mg./ 100 Cc.	No. of Cases	Mg./ 100 Cc.	No. of Cases	Mg./ 100 Cc.	No. of Cases	Mg./ 100 Cc.	No. of Cases	Mg./ 100 Cc.	No. of Cases	Mg./ 100 Cc.	No. of Cases	Mg./ 100 Cc.	No. of Cases
Atresia	11.1	(5)	10.4	(20)	10.9	(27)	10.9	(15)	12.1	(5)	15.1	(6)	17.8	(5)	22.4	(1)
Inspissated bile due to erythroblastosis fetalis	25.0	(26)	10.7	(11)	8.2	(4)	5.8	(3)	3.9	(1)	....	...	....	...	....	...
Inspissated bile of unknown cause	12.9	(9)	10.7	(20)	9.9	(20)	10.1	(11)	7.6	(8)	7.9	(5)	2.5	(1)	8.1	(3)
Miscellaneous	12.4	(1)	8.8	(5)	10.7	(5)	....	....	....	...	....	...	....	...	....	...

inspissated bile due to erythroblastosis showed high initial levels which fell to normal levels rapidly. The infants with inspissated bile of unknown cause had initial levels which were similar to those of infants with biliary atresia, but instead of rising, they tended to come down slowly. There were inadequate numbers of cases in the miscellaneous groups to permit interpretation.

Table 6 shows the initial bilirubin level of the infants when first admitted to the hospital, regardless of age. All infants with inspissated bile due to erythroblastosis fetalis had high bilirubin levels initially, while infants with biliary atresia and inspissated bile of unknown cause had low initial levels, with no significant differences between the two groups. There appeared to be no difference in initial levels between the cases of operative and inoperative biliary atresia or between the infants with inspissated bile who recovered and those in whom cirrhosis subsequently

TABLE 6.—*Total Serum Bilirubin on Admission to Hospital*

	Number Cases	Level, Mg./100 Cc.
Atresia	60	10.1
Inspissated bile due to erythroblastosis fetalis	23	25.5
Inspissated bile of unknown cause	24	11.3
Infectious hepatitis	3	17.6
Miscellaneous	5	10.3

TABLE 7.—*Changes of Individual Total Serum Bilirubin Levels*

Diagnosis	No. of Cases	Upward	Downward	Variable
Atresia	25	20	2	3
Inspissated bile due to erythroblastosis fetalis	13	0	13	0
Inspissated bile of unknown cause	16	0	6	10
Infectious hepatitis	3	0	3	0
Miscellaneous	3	1	1	1
Total	60	21	25	14

developed. The infants with infectious hepatitis had a slightly higher initial level than those with biliary atresia.

Table 7 shows the trend of bilirubin levels in the individual cases where two or more bilirubin levels were obtained serially several weeks apart. The trends have been defined as follows: (1) definitely upward or unchanged, (2) definitely downward, and (3) variable, characterized by rapid rise or fall of bilirubin levels over short intervals without any definite trends. Of the 25 infants with biliary atresia in whom serial determinations were obtained, 18 showed a definite upward trend and 2 others remained unchanged. In the same group, two infants showed a definite downward trend terminally. Three infants showed variable trends, of which two occurred terminally, and the third could not be explained.

All of the 13 infants with inspissated bile due to erythroblastosis showed a progressive downward trend. Of the 16 infants with inspissated bile of unknown cause, 6 showed a definite downward trend and recovered completely, while 10 others showed a variable pattern with rapid rise and fall of bilirubin levels. The variable pattern was seen both in the infants who subsequently recovered and in those in

whom cirrhosis developed. All of the infants with infectious hepatitis showed a downward trend, and the results of the miscellaneous cases were too few and variable to be interpreted.

COMMENT

On the basis of the above data, it would appear that the alkaline phosphatase and the liver function flocculation tests are of limited value in the differential diagnosis of obstructive jaundice in infancy. The values for the alkaline phosphatase obtained for the four types of obstructive jaundice are so similar that they cannot be distinguished from each other. Furthermore, the presence of normal values in infants with biliary atresia suggests that the test is unreliable in differentiating between obstructive jaundice and jaundice due to other causes in infancy.

The flocculation tests are consistently negative or borderline in all infants with biliary atresia, inspissated bile due to erythroblastosis, and inspissated bile of unknown cause and, therefore, cannot be used to differentiate one condition from another. They are useful, however, in separating obstructive jaundice from syphilis, galactosemia, infectious mononucleosis, and congenital cirrhosis, diseases which have been found to yield positive flocculation tests even in the very young infant.[3] Although positive flocculation tests are strongly suggestive of infectious hepatitis in infancy, negative flocculation tests do not necessarily rule out the disease in this age group.[4] Some infants with hepatitis proven by biopsy show negative flocculation tests for the first few months of life.[4]

The presence of bile in the stool definitely rules out biliary atresia, although its absence does not mean that the infant has that condition. Similarly, the presence of urobilinogen in the urine suggests incomplete obstruction, although its absence does not necessarily indicate biliary atresia.

The serum bilirubin level is the most useful test in differentiating between the causes of jaundice. Equal elevation of both the one-minute (direct) and indirect bilirubin levels indicates an obstructive jaundice and rules out the possibility of physiological jaundice,[5] erythroblastosis,[5] hemolytic anemias,[3] and sepsis, which all show elevation of primarily the indirect bilirubin level.

On the basis of the present studies of young infants with obstructive jaundice, a high initial bilirubin level in the 10 to 30 mg. per 100 cc. range suggests either inspissated bile due to erythroblastosis or hepatitis. A low initial bilirubin level in the 5 to 10 mg. per 100 cc. range suggests either biliary atresia or inspissated bile of unknown cause. If the levels are followed over a period of weeks, more definite diagnostic information is obtained. If the level shows a slow and steady upward trend with no variability, biliary atresia is strongly suggested. If the level shows a rapid downward trend, inspissated bile due to erythroblastosis or a hepatitis is suggested. If the level either shows a tendency to fall slowly or is highly variable, inspissated bile of unknown cause is suggested.

From the practical standpoint, liver function tests should be used together with blood grouping data and duodenal bile studies to decide between surgical exploration

3. Gellis, S. S., and Hsia, D. Y. Y.: Liver Function Tests in Infants and Children: A Review of 1,500 Cases, to be published.

4. (*a*) Gellis and Hsia.[3] (*b*) Weller, S. D. V.: Observations on Some Liver Function Tests in Infancy, Great Ormond St. J. **1**:26 (June) 1951.

5. Hsia, D. Y. Y.; Allen, F. H., Jr.; Diamond, L. K., and Gellis, S. S.: Serum Bilirubin Levels in the Newborn Infant: J. Pediat., to be published.

and medical management. If there is any doubt with regard to the diagnosis, it is felt that surgical exploration should be postponed until repeated examinations of stools and urine together with serial liver function tests including bilirubin tests have been performed. Since infants with biliary atresia amenable to surgery have been operated on as late as 8 months of age with complete recovery, a waiting period for the purpose of obtaining serial studies can be safely undertaken. Also, the poorer prognosis following surgery in the infants with inspissated bile of unknown cause would be additional indication for postponement. At the end of the period of study surgical exploration is indicated if (1) there has been consistently no bile in the stools and duodenal secretions, (2) there has been no urobilinogen in the urine, (3) the initial bilirubin level was low and has shown a slow progressive increase with no variability in pattern, (4) the flocculation tests are negative, and (5) there is no blood incompatibility. Surgery should be deferred longer if (1) there is bile in the stools or duodenal secretions even in small amounts, (2) urobilinogen is present in the urine in normal dilutions, (3) the initial bilirubin level was high and has shown a tendency to either fall or be highly variable with no definite upward trend, (4) the flocculation tests are positive, and (5) blood incompatibility suggesting erythroblastosis can be demonstrated. It has been found that differentiating between complete biliary atresia and incomplete obstruction is usually not difficult at the end of the waiting period.

SUMMARY

Liver function tests in 156 infants with obstructive jaundice have been evaluated.

Alkaline phosphatase and the flocculation tests are of no aid in differentiating between the various causes of obstruction. The flocculation tests may be helpful in distinguishing between obstructive jaundice and other neonatal conditions giving rise to jaundice.

The presence of bile in the stools and urobilinogen in the urine in normal amounts suggests incomplete obstruction.

Serial serum bilirubin levels are most useful in distinguishing the various causes of obstructive jaundice. A slowly rising trend suggests biliary atresia, a rapidly falling trend suggests inspissated bile due to erythroblastosis or may be indicative of a diffuse hepatitis, and a slowly falling or variable trend suggests inspissated bile of unknown cause.

A plan for deciding whether surgery should be undertaken in these infants is presented.

CROSSED LATERALITY IN CHILDREN

Report of Pediatric, Psychiatric, and Psychological Aspects of Three Cases

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FOR THE past two decades it has generally been conceded that the pediatric psychiatrist's proficiency has been greatly enhanced by his possession of a basic and sound knowledge of general pediatrics. Only recently, however, has the converse been recognized, namely, that a fundamental understanding of the basic principles of child psychiatry and pediatric psychology was essential to the pediatrician for the ideal conduct of his daily practice. In recent years two clinical factors have given impetus to the acceptance of a broader concept of the scope of pediatrics. The first has been the introduction and development of the psychosomatic aspects of certain disorders in children. The second has been a progressive awareness of the pediatrician as to the role he should play in the early recognition of disturbances of personality presented by his patients. In the diagnosis and treatment of particular cases the pediatrician has now become attuned to the necessity of coordinating his efforts, his knowledge, and his experience with those of the pediatric psychiatrist, the clinical psychologist, and, when the occasion demands it, the educational therapist. The limitations imposed by the specific training of these individuals are obvious, and the role each assumes in the diagnosis and management of a given case should be part of a collaborating plan of the team in order that the patient may receive the maximum benefit from their combined efforts. Of mutual interest to the pediatrician, the pediatric psychiatrist, the clinical psychologist, and the educational therapist, is crossed laterality, a challenging and intriguing human phenomenon. The presence of this condition in children may seriously interfere with the growth and development of their personalities and with their educational progress. This disorder frequently requires for diagnosis and proper management the integrated services and skills of each member of this professional group.

This report gives the results of an intensive study of three boys, all about 7 years of age, with crossed laterality. As will be illustrated in the individual case histories, each child presented severe emotional difficulties, disturbances of personality, problems of behavior, and retardation in school. The presence of crossed or mixed laterality in children as a factor contributing to the development of their personality, their emotional stability, and their progress in school is too seldom explored by pediatricians, psychologists, and educators. This failure of recognition may occasionally be due to a lack of familiarity with the nature of this disorder.

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It might therefore be expedient to describe this condition in some detail. Most children are right-handed and right-legged, some are left-handed and left-legged, and only the occasional child is truly ambilateral. In a similar way, one eye is dominant, usually on the same side as the handedness of the individual. To the same degree, one side of the brain is dominant, but on the side opposite to the preferred handedness of the child. Through correlating clinical and postmortem findings, Blau [1] has deduced that only one side of the brain seems to be all-important in the language process. The entire control of speech, reading, and writing is found to be centered in the same half of the brain which controls manual skill in the preferred hand. This side is known as the dominant hemisphere of the brain and is functionally superior to the opposite side. Thus, in dextrality cerebral dominance is left-sided, whereas in sinistrality it is right-sided. Blau states further that a very small injury in an appropriate area of the dominant hemisphere results in extensive loss of speech or of the ability to read or write, while an equal or even wider injury in the nondominant hemisphere is followed by no such language disorder. When the dominant eye of the child is opposite to the side of the preferred handedness, then that child is said to have crossed or mixed laterality. Thus, a child with crossed laterality may be right-eyed, left-handed, and usually left-legged, but occasionally right-legged, or he may be left-eyed, right-handed, and usually right-legged, but occasionally left-legged.

Crossed or mixed laterality, or crossed or mixed cerebral dominance, as the disturbance is frequently called, is not too uncommon in children. The basis for its occurrence and the nature of its physiology are not quite clear. In view of the fact that in dextrality per se cerebral dominance is left-sided, and in sinistrality per se it is right-sided, one can readily postulate that in crossed or mixed laterality cerebral dominance would be crossed or mixed. Therefore, it is not surprising to ascertain that the disorder frequently gives rise to certain types of educational disabilities and disturbances of personality. The diagnosis is established by specific psychological studies, which will be described later.

The treatment of crossed laterality involves two distinct aspects, namely, the emotional and the educational. Emotional adjustment may be accomplished either by the pediatrician, if he possesses the necessary training and experience, or by the pediatric psychiatrist. At times this emotional adjustment must be made before educational therapy can be instituted. Each case must be individually evaluated. There can be no absolute or definite method of treatment for emotional disabilities and disturbances of personality because of the wide variation in the reactions of each child to the given stimulus or stimuli involved and to the therapeutic modalities employed. These children, as a rule, do not respond to ordinary methods of education, so they frequently require technical remedial measures by an educational therapist. The utilization simultaneously of visual, motor, tactile, and auditory senses, with the occasional addition of phonetic and kinesthetic cues, is essential in educating the patient. The cooperation of the child's parents as well as his teachers in school is important for the successful management of the

1. Blau, A.: The Master Hand: A Study of the Origin and Meaning of Right and Left Sidedness and Its Relation to Personality and Language, Research Monographs No. 5, New York, American Orthopsychiatric Association, 1946.

case. Though most children outgrow the symptoms of crossed or mixed laterality spontaneously at about 12 years of age, unfortunately in some of these untreated boys and girls fixed and irreversible disorders of personality may develop. The necessity of early diagnosis and the institution of proper corrective procedures thus become apparent.

REPORT OF CASES

Case 1.—G. B., 6⅝ years old, the older of two children, was brought to the pediatrician with the complaint of inattentiveness, inability to concentrate, difficulty of learning in school, and alterations in his personality. His mother stated that she still had to feed him and that he always had a sensitive temperament. Until recently, his father suffered from migraine and readily went into temper tantrums. During these episodes, he would berate his son at the slightest provocation. The boy slept in the same bedroom as an uncle who is a borderline mongoloid. Shortly after entering school, the child became inattentive to his parents and to his teachers; the latter complained of his lack of effort, his apparent unwillingness to concentrate on his work, and his inability to learn how to read or to spell. Within recent months, he had become enuretic, and, at times, he had soiled himself either in school or on the way home from school. He also became a poor sleeper and eater. His parents noted a distinct change in his general behavior. The findings on examination of the child were within normal limits from the physical and neurological point of view. He appeared very alert, cooperative, and agreeable. He possessed an excellent vocabulary. He was interested chiefly in the mechanics of electric trains and diesel engines. Because of the disturbance in his environment, the alterations in his behavior, and his difficulty in learning, a psychometric test was recommended, the results of which are tabulated later. Measures were instituted to adjust his environment and to correct his abnormal behavior. Remedial therapy by an educational therapist was also begun. Within a period of two months the boy's behavior has responded remarkably to treatment. He has not soiled himself, and his enuresis has completely disappeared. His sleeping and eating habits are better, and he has become more attentive to his parents and to his teacher. His writing reversals have disappeared, and his work in school has been progressing satisfactorily.

Case 2.—A. P., 7 years old, an only child, was brought to the pediatrician with the complaint of inattention, lack of concentration, and difficulty of learning in school and, in addition, severe disturbances of behavior both at home and in school. He was taken into the family one day after birth and was legally adopted later. The boy's emotional development apparently followed a normal pattern up to 3 years of age, when his parents had a serious quarrel. His mother was compelled to go to work, and during the day the child was cared for by a relative. He soon became disobedient, hyperactive, and difficult to manage. At 4 years of age he was sent to nursery school, where he spent the entire day. At 6 years of age he was entered in public school, and after his session there he was picked up and taken to nursery school, where he stayed until evening. At about this time his father failed in business, and his parents had a second serious quarrel. This parental malrelationship so adversely affected the boy that he became totally unmanageable, defiant, aggressive, and mischievous, fighting at the slightest provocation. He became careless about his dress and his personal hygiene. He developed both diurnal and nocturnal enuresis. He also became a poor sleeper. At school he was very inattentive and manifested no interest in his work. He would run around the classroom or outside into the corridor and other classrooms and generally disrupt the entire floor. Efforts of the principal and the teachers to reason with him proved of no avail. The boy was transferred to a parochial school, where he continued the same pattern of behavior. A retransfer to a second public school provided no change in his reactions, and it became necessary to enroll him in a private school specializing in the training and education of children with behavior problems. In the preceding two years he had been seen by several pediatricians, who advised the parents that the child would probably outgrow this disorder of behavior spontaneously. On one occasion hospitalization was recommended for observation and treatment. Examination of the child for this present study revealed a hyperactive and mentally alert boy. At times his actions appeared sudden, impulsive, and unprovoked. He possessed an excellent vocabulary and was quite

cooperative. His pupils were dilated but equal and reacted to light and accommodation. His eyegrounds were normal. He held his muscles tense, and his reflexes, both deep and superficial, were exaggerated. There was a marked response to sensory stimulation. The findings on physical and neurological examination were otherwise within normal limits. Because of his disturbing environmental history, his abnormal behavior, and his difficulty in learning at school, he was given a psychometric test. The results of this psychological study are summarized later. In treating this boy adequate measures were first instituted to adjust his environment to one of happiness, contentment, and security and to change the attitude of his parents toward each other and toward the child. In the interim, both psychiatric and specific remedial educational therapy were given. Within a few months the boy began to lose his feeling of insecurity and doubt. His enuresis disappeared, and he now slept soundly. His restlessness and hyperactivity decreased, and he was more attentive in school. He was no longer defiant and pugnacious, and he now began to submit to parental discipline. In addition, he has made almost 18 months of progress in his work at school.

Case 3.—S. S., 7 years old, was brought to the pediatrician with the complaint of inattention, lack of concentration, difficulty in learning in school, and severe problems of behavior. The boy was legally adopted at an early age. His father, 49 years of age, has hypertension and temper tantrums and frequently quarrels with his wife, who is 12 years his junior. These quarrels would cease only when the boy became excited and yelled at them to stop fighting. The parents were very solicitous and anxious about their adopted son's welfare. The boy did well physically and emotionally up to 14 months of age, when he began to rock back and forth ceaselessly in his crib during the night. This habit had continued, though it had abated somewhat in intensity in the past year. He was still a poor sleeper and insisted on going to bed with one of his parents. He must have a light on in his bedroom. He walked at 18 months and talked at 2 years of age. Urination and defecation were controlled at 2 years of age. Since early childhood he had been defiant, aggressive, unmanageable, and demanding attention. This abnormal behavior had been progressive in intensity. He had been doing very poorly in school, being inattentive and apparently unable to concentrate. He seemed to lack confidence in his ability to do anything of an educational nature. His mind was always flitting from one subject to another, and he appeared indifferent to his educational inadequacies. He did not play with any children except his cousin. He was quite upset when he was told by his mother that he was adopted. In the preceding five years he had been seen by several pediatricians, psychiatrists, and neurologists. He was also taken to several agencies interested in child behavior. The diagnosis of childhood schizophrenia was seriously entertained, and hospitalization was repeatedly recommended. At the onset of his physical examination the boy was excited and extremely restless. He vigorously sucked his thumb and, grasping his mother's hand, demanded that he be taken home. After 20 minutes of general play the parents were dismissed, and the boy became cooperative and readily submitted to examination. He was generally tense, and all of his reflexes were exaggerated. There was a marked response to all sensory stimulation. His pupils were dilated and slightly unequal, but reacted to light and accommodation. His eyegrounds were normal. His heart rate was 104. The findings on physical and neurological examination otherwise were essentially within normal limits. From his history and physical examination it was apparent that the boy presented a marked emotional disturbance based primarily on a feeling of insecurity and doubt. Fear also seemed to be a dominating emotion. Probably as a defensive mechanism he had regressed to many infantile traits, such as thumbsucking, grasping his mother's hand, and crying when confronted with a difficult situation. His mother seemed to be shielding an inner rejection of the child and to have a consequent feeling of guilt. The boy apparently sensed this and resented her attitude. The child revealed a deep insecurity with a compensatory hostility, aggressiveness, and infantile regression. The results of a psychometric test administered to the child are summarized later. The decision was made to hold his remedial educational therapy in abeyance and to concentrate on stabilizing his behavior. Such a program called for the adjusting of his environment to one of contentment, happiness, and security and for the modifying of the attitude of his parents, not only toward the child, but also toward each other. Simultaneously the child's attitude toward his parents had to be changed to one of love and trust. The elements of fear had to be eradicated, and

he was made to feel more secure within his immediate family circle. Satisfactory progress has been attained within a short period of time. There has been a marked improvement in his behavior. His rocking in bed has disappeared completely. He now goes to bed alone, and the light in his bedroom is extinguished as soon as he retires.

PSYCHOLOGICAL STUDIES

In order to establish definitely the presence of crossed or mixed laterality, the three patients were examined psychologically. The testing program included the following determinations: (1) laterality, (2) intellectual capacity and efficiency, (3) achievement in school, (4) visual-motor integration, and (5) projection of a human figure by means of pencil drawings.

The patients' tendencies in laterality were determined by the following testing procedure: The preferred handedness was established by having the child (*a*) throw and catch a ball, (*b*) twirl his thumbs, (*c*) clap his hands, and, for finer manipulation, (*d*) write, (*e*) string beads, and (*f*) unravel a knot. Some of these and other tests, such as the ambigraph, are described by Jastak.[2] Dominance of the eye was ascertained by utilizing the Parsons [3] manuscope variation. The principle of this test is to observe the eye preference of the patient, though he is unaware

TABLE 1.—*Summary of Results of Laterality Tests*

	Handedness		Dominant Eye		Leggedness	
Patient	Right	Left	Right	Left	Right	Left
G. B.	..	X	X	..	X	..
A. P.	..	X	X	..	..	X
S. S.	X	..	..	X	..	X

that he is sighting with one eye. The test is made by holding at arm's length a cardboard or a piece of paper with a 1-in. hole in it. The subject looks through the hole at an object some distance away, and the examiner notes which eye is being used for sighting. Several trials are given. Another practical test is to have the child look through a microscope or small telescope and to note which eye he prefers for sighting. According to Parsons,[3] dominance of the eye is not dependent on acuity of vision, because usually acuity is equal in both eyes and occasionally it may be even less in the dominant eye. Ocular dominance is a preferred function of the individual in monocular sighting or in ocular fixation. Therefore, mild disturbances of vision, such as minor astigmatism or small errors of refraction, do not as a rule play a role in determining ocular dominance. Preferred leggedness can be established by having the child kick an object on the floor or by having him stand on one foot. The results of the preferred laterality tests are summarized in Table 1.

The interesting observation from a clinical point of view is that crossed or mixed laterality was present in all three patients. Two of these boys revealed left-hand preference and right-eye dominance, but one showed right-leg preference, whereas the other had left-leggedness. The third child was right-handed, with left-eye dominance and left-leggedness.

2. Jastak, J.: The Ambigraph Laterality Test, J. Appl. Psychol. **23**:473-487, 1939.

3. Parsons, B. S.: Lefthandedness: A New Interpretation, New York, The Macmillan Company, 1924.

The Terman-Merrill 1937 Revised Stanford-Binet Intelligence Scale, Form L,[4] was employed to establish the patients' mental ages and their intelligence quotients. Two of the children, A. P. and S. S., showed I. Q.'s within the normal range of 90 to 110. The third patient, G. B., established an I. Q. of 129, which placed him in the category of very superior intelligence. The scores on the vocabulary subtest of A. P. and G. B. merited them a vocabulary age of one year beyond their chronological age and of S. S. three years beyond his chronological age. The basal age for A. P. was established in year five and in S. S. and G. B. in year six. The subtest in year seven, in which all three children failed consistently, required the copying of the figure of a diamond 2¾ in. long and 1½ in. wide. The best of

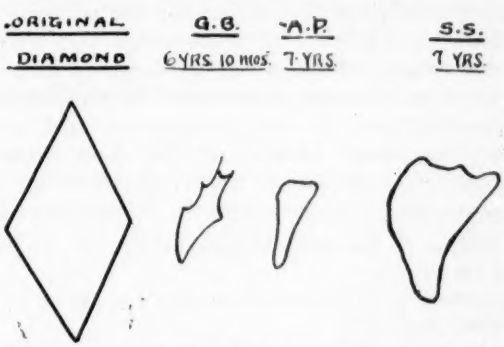

Fig. 1.—Patients' reproductions of a diamond figure (Subtest Year 7, Stanford-Binet, L.), Third trial. Note the irregularities and the distortions of the reproductions both as to size and shape as well as to angulation in comparison with the original diamond.

TABLE 2.—*Summary of Results Obtained by Patients on the Terman-Merrill Revised Stanford-Binet Intelligence Scale, Form L*

Patient	Chronological Age, Yr.	Mental Age, Yr.	Intelligence Quotient	Vocabulary Subtest, Yr.
G. B.	6 5/6	8 5/6	129	8
A. P.	7	6 2/3	95	8
S. S.	7	7 1/3	105	10

three efforts made by these children are reproduced in Figure 1. The reproduced diagrams are all small and very irregular. It is significant that failure in this identical subtest occurred in all three patients and was probably a factor in depressing their I. Q.'s. Terman [4] states:

> It is most puzzling that a child who can draw a square quite acceptably may be unable to draw a diamond. Success with either hinges upon the ability of the child to break up the unitary character of the perceptual experience in the reproduction of the figure, and difficulties in execution are involved when the parts have been perceived not as parts but features of a whole.

The results of the tests for intellectual capacity and efficiency are indicated in Table 2.

4. Terman, L. M., and Merrill, M. A.: Measuring Intelligence: A Guide to the Administration of the New Revised Stanford-Binet Tests of Intelligence, Boston, Houghton Mifflin Company, 1937.

Jastak's [5] Wide Range Achievement Test was utilized to obtain a rating of the patients' educational achievement in the fundamental school subjects of reading, spelling, and arithmetic. The reading test is essentially one of pronouncing words rather than one of comprehension. In the word-reading test the child's skill is assayed without the interference of context and comprehension. The correlation of the word-reading test with the New Stanford Paragraph Test is 0.94. The spelling test also includes the prespelling level of achievement. Thus, the child who is unable to spell the simplest word is asked to write his name or, if he is unable to do so, to write or print as many letters as he can. This simple performance is a fairly accurate measure of the development of coordinations and symbol perceptions needed for normal progress in written spelling. The arithmetic test contains 79 computation problems representing the fundamental processes of addition, subtraction, multiplication, and division. If the child is unable to solve any of the computational problems because his achievement level is below the level of the second grade, an oral test is administered to determine the development of his number concept at the preschool level. In order to comprehend and interpret the results obtained on the Wide Range Achievement Test, it is essential to know the child's chronological age, his mental age, and his educational age achievement level in each of the tool subjects. The age data provide the following ratios or quotients:

1. Intelligence Quotient (I. Q.)—defined as the mental age (M. A.) × 100 divided by the chronological age (C. A.).
2. Educational Quotient (E. Q.)—defined as the educational age (E. A.) × 100 divided by the chronological age (C. A.).
3. Accomplishment Quotient (A. Q.)—defined as the educational age (E. A.) × 100 divided by the mental age (M. A.).

The I. Q. categorizes the child's innate intellectual status. The educational quotient reveals whether a child is educationally proficient or backward or, in other words, whether or not he is successfully competing with other pupils of his chronological age. The accomplishment quotient discloses whether or not a pupil's accomplishment in a school subject is in consonance with his intellectual ability. If his accomplishment quotient in a subject is much below his I. Q. the pupil is said to be retarded in that subject. In this instance, the pupil is being evaluated on the basis of his intellect and is not in competition with other children. Table 3 shows the educational and accomplishment quotients obtained from the three children included in this study.

An inspection of Table 3 reveals that patient G. B. (C. A., 6⅝ years; M. A., 8⅝ years, and I. Q., 129) is proficient in his school work, i. e., he is keeping up with the grade work of children of his own age, but he is seriously retarded in all three subjects. In other words, he is not accomplishing as much as is expected in view of his M. A. of 8⅝ years. His poorest effort is in spelling. Confusional trends and reversals were noted in his attempt to write words such as "in" and "go," and he did mirror writing with numerical figures. These are illustrated in Figure 2. His errors in reading were mostly ideational, as he employed guesses and supplements. Thus for "milk" he read "make," for "tree," "three," for "was," "has," for "eat," "cat," and for "now," how." The results of this achievement

5. Jastak, J.: Manual of Wide Range Achievement Test, Wilmington, Del., Charles L. Story Co., 1941.

test demonstrate the case of a boy with very superior intelligence who is retarded educationally. His retardation appears to be tied up with his crossed laterality and his confusional trends in writing and ideational errors in reading appear to be associated with his ignorance of the strephic and phonic elements and his disturbed behavior in school as well as his unfavorable emotional status at home. A. P. (C. A., 7 years; M. A., 6⅔ years, and I. Q., 95) is moderately backward educationally in reading and arithmetic, but he definitely lacks proficiency in spelling. In one year of schooling he has managed to learn how to write his first name. In Figure 2 he demonstrates reversals in writing, thus disclosing the confusional effect of mixed dominance in his attempts at learning how to write. His accomplishment in reading and arithmetic approaches the normal limits for his M. A., but he is definitely retarded in spelling and writing. S. S. (C. A., 7 years; M. A., 7⅓ years, and I. Q., 105) is educationally backward in all three subjects. After one year of schooling he has learned how to recognize only one letter of the alphabet,

TABLE 3.—*Results Obtained by Patients on Jastak's Wide Range Achievement Test*

Subject	Grade	Grade Equivalent	Age Equivalent, Yr.	Educational Quotient	Accomplishment Quotient
		Patient G. B.			
Reading	1.7	1B	6 2/3	98	75
Spelling	1.3	1A	6 1/3	93	72
Arithmetic	2.3	2A	7 1/3	107	83
		Patient A. P.			
Reading	1.2	1A	6 1/6	80	93
Spelling	0.7	..	5 2/3	81	85
Arithmetic	1.4	1A	6 5/12	92	96
		Patient S. S.			
Reading	0.3	..	5 1/3	75	73
Spelling	1.0	1A	6	86	82
Arithmetic	0.7	..	5 2/3	81	77

the letter "S," which begins his first and second name. In writing his full name he omitted the letter "e" and reversed the letter "p" in his first name and was unable to complete his second name. He did marked mirror writing with figures. These reversals are illustrated in Figure 2. In the arithmetic test he obtained credit for counting up to 15. This case illustrates a boy with above average intelligence in whom serious educational difficulties have arisen which, undoubtedly, have exacerbated his disturbance of behavior and his emotional state. The presence of mixed laterality with concomitant failure to learn how to read, spell, and write cannot be ruled out as a factor in further undermining the child's personality growth and adjustment.

The Bender Visual-Motor Gestalt Test was employed to evaluate the maturation level of the patients' visual-motor gestalt function. Bender [6] states that the test is an experimental device, "to explore retardation, regression, loss of function, and organic brain defects in both children and adults and to explore personality deviations, especially where there are regressive phenomena." The test consists

6. Bender, L.: A Visual Motor Gestalt Test and Its Clinical Use, Research Monograph Series No. 3, New York, American Orthopsychiatric Association, 1938.

of nine geometric forms drawn in black on a white piece of cardboard about the size of a postcard. Bender [7] gives the following directions for administering the test: "Here are some figures or designs for you to copy. Just copy them as you see them." The interpretation of the test results depends on how the child perceives the stimulus of the gestalt pattern on the card, what it means to him, and what he does to it in his attempts to reproduce it on paper. The criteria for the evaluation of the test responses are offered by Bender [7] and by Halpern in her article in Anderson and Anderson's book on projective techniques.[8]

The reproductions of the gestalt patterns by the three patients revealed significant deviations, and several of the drawings of the gestalten are shown in Figure 3. Each gestalt pattern has a specific designation, such as "Card A" or "Card 5." G. B.'s reproductions of the gestalten disclose the following gross motor disturbances and disorders in perceptual organization: In Card A there is displacement and overlapping of the unitary figures. In Card 4 separation of parts is noted with

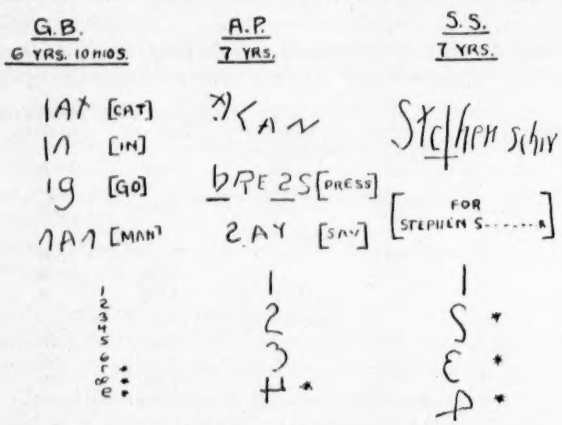

Fig. 2.—Reversals in handwriting. Note the micrographia of G. B. The mirror writing of the numerical figures is signified by asterisks.

micrographia. In Card 5 the dots have been replaced by continuous lines suggesting a reluctance on the part of the patient to exert himself by drawing dots, which is a time- and effort-consuming task. In Card 7 marked difficulty with angulations is present with resultant loss of the gestalt pattern. In Card 8 the horizontal figure is completely rotated on its axis, revealing a derangement in spatial orientation. A. P.'s reproductions of the gestalten show the following abnormalities: In Card A there is overlapping of the unitary parts of the gestalten with loss of pattern, the square figure becoming a diamond with poorly joined and open angles. In Card 4 there is displacement and some reversal of the bell-shaped figure with serious distortion in an attempt to synthesize the gestalt. Card 5 shows primitivization and oversimplification of the dots into continuous lines with the additional

7. Bender, L.: Instructions for the Use of the Visual Motor Gestalt Test, Monograph, American Orthopsychiatric Association, 1946.

8. Anderson, H. H., and Anderson, G. L., editors: An Introduction to Projective Techniques, and Other Devices for Understanding the Dynamics of Human Behavior, New York, Prentice-Hall, Inc., 1951.

lines with the additional disturbance of reversal. The rejection of the more difficult task of drawing dots and the rotational disturbance are strongly suggestive of an organic disorder in the visual-motor integrative area of the brain. In Card 7 there is separation of parts with marked evidence of emotional excitement in attempting to draw the angles. In Card 8 separation of parts with micrographia denotes an inability to control and inhibit impulses easily. S. S.'s reproductions of the gestalten contain serious abnormalities and deviations. In Card A the fragmentation and wide separation of the entities comprising the gestalt suggest the presence of severe motor difficulties and organizational shortcomings. In Card 4 the reproduction is excessively enlarged, denoting lack of inhibition. The fragmen-

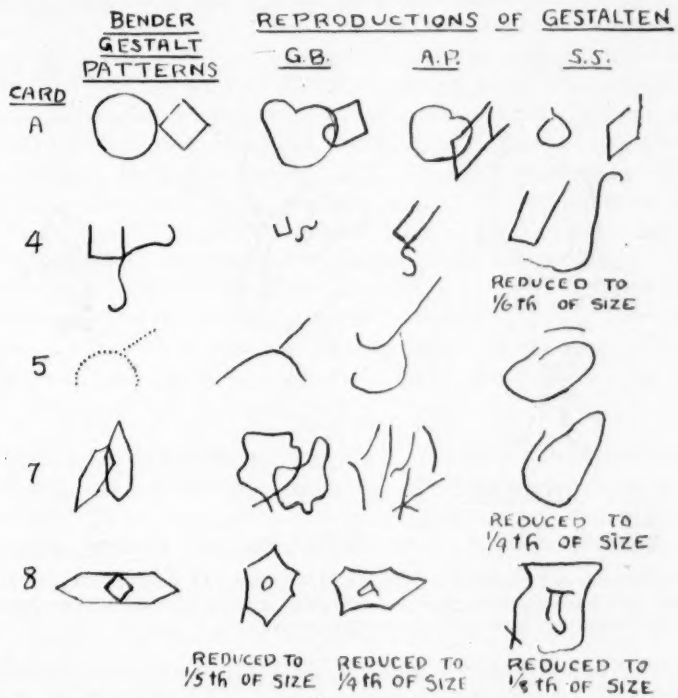

Fig. 3.—Reproductions of the Bender Visual-Motor Gestalt Patterns. Some of the reproductions are considerably reduced in size. Observe the marked distortions, indicating (1) confusion in the visual motor integration area of the brain, (2) spatial disorientation, (3) regressive tendencies, and (4) disruption of personality.

tation and severe distortion of the bell-shaped figure indicate a fundamental disturbance in spatial orientation. In Card 5 there is almost complete loss of the gestalt as a result of primitivization and oversimplification. In Card 7 the difficulty in handling angulations and the concomitant emotional disturbance is shown by his drawing of only one of the entities of the pattern with marked exaggeration. In Card 8 the pattern was drawn greatly enlarged and at a 90-degree reversal with separation of parts. The heightened emotional upset and severe motor difficulties are clearly in evidence.

In the gestalt test, which was standardized as a performance test for children, the average child 7 years of age is able to reproduce with accuracy Cards A and 5 and for the other patterns recognizable and integrated reproductions. In the three reported cases of crossed laterality the drawings of the gestalten by these children reveal several pathognomonic signs indicating the presence of motor disturbances, regressive tendencies, and disruption of personality. It is interesting to note that two patients, G. B. and A. P., are left-handed, and that all three children have crossed laterality. The drawings of the gestalt patterns of each child show gross abnormalities and distortions.

The Goodenough Intelligence ("Draw-a-Man") Test [9] was utilized to obtain, in the words of Gesell,[10] "the child's concept of a man and what he has learned

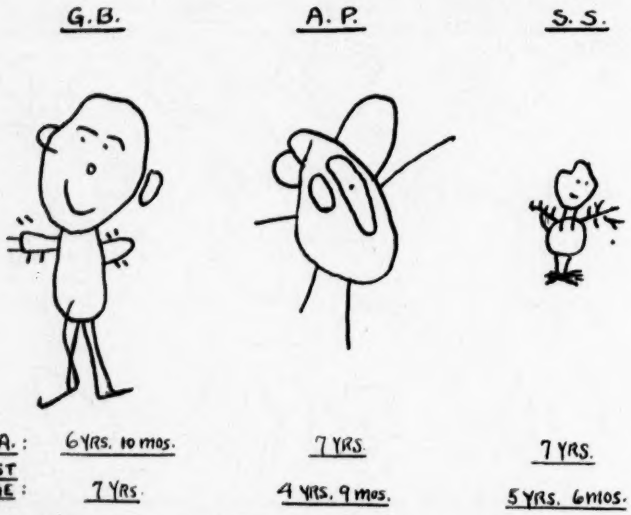

Fig. 4.—Goodenough Intelligence ("Draw-a-Man") Test. All three drawings of the human figure point to (1) inadequate perception of the human in space, (2) some organic concomitants, and (3) deterioration or regression of personality structure.

to draw that, to him, looks like a man or some part of a man." In addition, the human figure drawings of these children served as a projective technic. The interpretations of unusual details shown in human figure drawings, as noted by Machover in an article in Anderson and Anderson's book on projective techniques,[8] are described in the analysis of the patients' endeavors. The reproductions of the patients' drawings of the human figure are shown in Figure 4.

G. B.'s (I. Q., 129) drawing strikingly shows exaggeration of the head, which is oddly shaped and twisted towards the right with the right ear overlapping and the left ear floating in the air. The fingers stick out as six wavy lines on the right arm and four on the left. The trunk is flattened out on othe lower right side. The

9. Goodenough, F. L.: Measurement of Intelligence by Drawings, New York, World Book Company, 1926.

10. Gesell, A.: The First 5 Years of Life, New York, Harper & Brothers, 1940.

right leg is larger than the left. The distortions in this human figure drawing appear to be on the right side of the body, with a large head almost half the size of the body, suggesting deemphasis of body control and coordination and the presence of an organic brain condition relating to laterality. A. P.'s human figure drawing consisted of a large distorted head with the omission of the trunk. He included details in the head to merit a Goodenough M. A. of 4¼ years. The head is tilted towards the right, coming to a point, and open at the top. The right eye and ear are much smaller than the left. The arms, shown as straight lines, extend from the head in the region of the eyes. Two straight lines extending downward from the head apparently represent legs. The oddly-shaped head is strongly suggestive of some disturbance in brain structure or function. The uneven size of the eyes and ears, the left being larger than the right, points to social maladjustment concerned with seeing and hearing. There is a conflict between what he should see and hear and what he should not see and hear. The omission of the trunk and the misplaced arms and legs indicate an infantile approach to body control and concept of the human figure. S. S.'s human figure was drawn hurriedly and protestingly. The human figure is small. There is some awareness of differentiated parts such as head, neck, trunk, and limbs. The head, however, contains a dent in the upper right side. The facial features are not clearly demarcated. The loci of most serious disturbances may be seen in the hands and feet. He drew straight lines for fingers without taking the trouble to count them, and he scribbled in the region of the feet. An interpretation of his drawing reveals a lack of body control and integration with deep concern or displeasure in body function concerned with the use of hands and feet. Brain dysfunction seems to be in evidence.

COMMENT

Though the problem of laterality in general has received considerable attention from neurologists, psychiatrists, psychologists, and educators, the subject of crossed or mixed laterality has not apparently stimulated sufficient interest for its exploration as a factor in producing personality disorders and learning difficulties. It is not within the scope of this presentation to review and comment on the many controversial theoretical and philosophical discussions on laterality appearing in the literature. It might be pertinent, however, to select a few excerpts from the literature to illustrate these conflicts. Blau [1] states that reading reversals and mirror writing are the results of developmental anomalies or habituated fixations in an erroneous choice of eye direction gaze, and these lead to faulty perception of symbol structure and are factors in the development of preferred laterality of the yes. The acceptance of the concept of sinistrality and of the sinistral choice of eye direction gaze as a cause of language disorders has been rejected by many authors. For example, Rheinberger, Karlin, and Berman [11] have demontrated that there was no significant difference between stuttering and nonstuttering boys from the point of view of laterality tendencies and the encephalographic patterns.

For decades the controversy has existed as to the advisability of changing the apparent dominant laterality of a child. One school of thought strongly advocates

11. Rheinberger, M. B.; Karlin, I. W., and Berman, A. B.: Electroencephalographic and Laterality Studies of Stuttering and Nonstuttering Children, Nerv. Child **2**:117-133, 1943.

just such a procedure. Their belief is based on the premise that cerebral dominance is a psychosomatic or acquired trait determined by function, and that the choice of left or right dominance is decided by experience and learning. This learning is impressed in the corresponding hemisphere and sets the path for the other features of dominance, particularly those of the language centers. In contrast to this view, a second school of thought opposes such a drastic step as altering a child's preferred laterality. Hume [12] states that there is a chance that interference with a child's natural tendency to use one hand or the other may make him nervous or give rise to speech disorders or to other evidences of emotional instability. It is therefore wiser, according to Hume, not to run the risk of creating a nervous imbalance through trying to make a right-hander out of a left-hander or vice versa.

The three children included in this study were brought to the pediatrician with almost identical educational complaints, though their behavior patterns differed. Each child presented marked learning difficulties. The first child, G. B., was fairly proficient in his school work but was educationally retarded in view of his superior intelligence. The two adopted children, A. P. and S. S., were educationally deficient and retarded in reading, spelling, and arithmetic. All three were reported by their teachers for inattention, for lack of concentration and application, and as a disturbing influence in the classroom. The educational retardation occurred in all three patients in spite of the fact that the first boy had an exceptionally high I. Q., and the other two boys had average I. Q.'s. In addition, each child presented symptoms of a disturbance of personality. The first child, G. B., did not develop any evidence of an emotional disorder until after he had entered school. The deduction may therefore be made that crossed laterality was a factor in producing his emotional disability. The two adopted boys, A. P. and S. S., displayed signs of emotional tension and disturbances of personality rather early in life, though their symptoms of disorder of behavior were exaggerated after they had entered school. Here too crossed laterality undoubtedly accentuated their emotional instability and educational discrepancies. Both of these boys were considered as preschizoid by some, and hospitalization had been recommended. Each of these children had numerous examinations, but the presence of crossed laterality had not been ascertained. The suggestion is offered that children who have learning difficulties be tested for preferred laterality as routine neurological and psychological procedure.

SUMMARY

Three cases of crossed or mixed laterality or crossed or mixed cerebral dominance are presented. An inspection of the results of the psychometric program administered to the three patients in this study discloses a consistent pattern of disturbance in their visual-motor integration, perception, and spatial orientation. The presence of crossed laterality was indicated in all three cases. In the Terman-Merrill Revised Stanford-Binet Intelligence Test, Form L, marked distortion of proportion and loss of angulation was observed in their failure to copy correctly the figure of a diamond. On the Wide Range Achievement Test reversals in writing were seen. In addition, serious educational deficiencies in the tool subjects of reading, spelling, and arithmetic were noted. The results of the Bender Visual-

12. Hume, A. C.: Helping the Left-Handed Child, J. Exceptional Child. **17**:201-203, 1951.

Motor Gestalt Test showed serious pathognomonic signs of disturbances in the visual-motor integration area of the brain. The three children also revealed evidence of a disturbed emotional state and deterioration of personality. Their drawings of the human figure pointed up to inadequate perception of the human in space, some organic concomitants, and deterioration or regression of personality structure.

Crossed laterality is not too uncommon in children, and the pediatrician should be alert to recognize the condition. Children who manifest difficulties in learning should have the status of their intelligence and their educational attainment professionally assessed. The possiblity of crossed laterality as a factor for their emotional and educational disturbances should be entertained, and appropriate psychometric tests as well as a complete pediatric, neurological, and psychiatric examination should be performed. On the basis of the present study it is suggested that appropriate psychiatric and remedial educational therapy be the method of choice in the proper management of crossed laterality.

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COCCIDIOIDOMYCOSIS OF BONE IN CHILDREN

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OSSEOUS coccidioidomycosis in children occurs as a dissemination of the disease. A search in the *Quarterly Cumulative Index Medicus* revealed no specific publications on the subject of coccidioidomycosis of the bone in children. There is unquestionably a prevalence of misinformation regarding the morbidity and mortality of this disease. In our opinion, it is a chronic self-limiting disease which rarely ends fatally unless there is generalized systemic involvement in addition to the osseous involvement. Caffey [1] summed up the subject briefly under the

TABLE 1.—*Data on Five Patients with Coccidioidomycosis Treated 1909-1930**

Patient	Age, Yr.	Bone Lesion	Laboratory	Comment
E. D.	2	Right ankle	Autopsy findings	Died
J. G.	9	Fingers	Autopsy findings	Died
W. B.	1 1/6	Hand bones	Laboratory test positive	Died
F. J.	2½	Skull; os calcis; vertebrae	Laboratory tests positive	Died
M. P.	8	4th metatarsal	Laboratory test positive	Recovered

* These data were supplied by Myrnie A. Gifford, M.D.

heading "Mycotic Osteomyelitis" by stating that the cranium was sometimes involved in the rarer chronic infections, such as actinomycosis, blastomycosis, and coccidioidomycosis.

One of the most traveled highways in the United States is through the heart of an endemic area for coccidioidomycosis. As a result of this study and from personal experiences, we feel that this form of disseminated coccidioidomycosis occurs relatively frequently in endemic areas and is overlooked elsewhere, primarily because of a lack of vacational and previous residential history.

Surgical Resident, Long Beach Veterans Administration Hospital (Dr. Dykes); Attending Radiologist, Kern General Hospital (Dr. Birsner).

1. Caffey, J. P.: Pediatric X-Ray Diagnosis: A Textbook for Students and Practitioners of Pediatrics, Surgery, and Radiology, Chicago, Year Book Publishers Inc., 1945.

TABLE 2.—*Clinical Data on Twenty-One Cases of Coccidioidomycosis Observed 1930-1950*

Case	Age, Yr.	Sex	Race or Nationality	Chief Complaint	Duration	Bone Lesion	Other Lesions	Laboratory Data	Comment
1	5/12	F	Mexican	Pain and swelling of toe	2 wk.	Middle phalanx rt. 4th toe	Minimal infiltration apex of lung	Skin test equivocal; serologic reactions, and culture +	.....
2	1	M	Mexican	Nodules of scalp	.....	Rt. clavicle; frontal bone; 1st & 5th metatarsal; tibia, & lt. humerus & mandible	Lung at age of 1 mo.	Skin test, serologic reactions, smear, and culture +	Soft tissue mass developed over left mandible, with breakdown 1 mo. following discovery of underlying osteolytic lesion
3	6	M	Negro	Pain and swelling of nose and jaw	3½ yr.	Rt. and lt. mandible, nasal bone	Lymph nodes	Skin test, serologic reactions, smear, and culture +	Coccidioidal spherules found in lymph node biopsy, but not in bone biopsy.
4	6/12	F	White	Swollen finger	5 mo.	Middle phalanx rt. 4th finger	Pulmonary involvement	Skin test, serologic reactions, smear, and culture +	Coccidioidal pneumonia at age of 2 wk., followed in 2 wk. by osseous lesion
5	4	F	Negro	Swelling around right eye	3 wk.	Rt. maxillary bone and zygomatic process	Pulmonary involvement	Skin test equivocal; serologic reactions, and culture +	Accompanying soft tissue abscess
6	2½	M	Mexican	Bumps on head for 1 mo.; fever for 3 wk.	1 mo.	Frontal bone	.....	Skin test, serologic reactions, and culture +	.....
7	5	F	Mexican	Swollen left ankle	3 wk.	Lt. os calcis	Minimal infiltration base of lungs	Skin test, serologic reactions, smear, and culture +	.....
8	10	M	Negro	Injury to back with swelling and abscess	1 mo.	Rt. 10th rib	.....	Skin test, serologic reactions, and smear +	.....
9	9	F	Negro	.....	7 yr.	Multiple bone involvement, esp. T 12 and L 1	.....	Skin test +	.....
10	7	F	Negro	Back pain	1 wk.	Spinous process L 1	Lung 1 mo. later	Skin test, serologic reactions, smear, and culture +	.....
11	2	F	Negro	Swelling and pain, back of head	3 wk.	Occipital bone	.....	Skin test, serologic reactions, smear, and culture +	Lumbar abscess developed; died with disseminated coccidioidomycosis
12	10	M	Negro	Acute illness with stiff neck	2 mo.	Lt. 5th rib, T 3, 4 and 8	Pulmonary involvement	Skin test equivocal; serologic reactions, smear, and culture +	Injury to head prior to swelling and abscess formation Died with disseminated coccidioidomycosis
13	10	M	Mexican	Abscess, right ankle	7 mo.	Rt. os calcis	.....	Guinea pig inoculation +	Fracture of rt. ankle age 3 yr.; for 6 mo. remained swollen and drained; 7 mo. prior to entrance abscess developed same site
14	13	M	Negro	Abscess over clavicle	3 wk.	Rt. clavicle	.....	Smear and culture +	History of injury to clavicle; also swelling over left extremity; malleolus, but no x-ray taken
15	8½	M	Negro	Swelling, redness, and tenderness, right hand	1 wk.	Rt. 3d metacarpal, skull	.....	Smear and culture +	.....
16	4	F	Mexican	Abscess, back	3 mo.	T 12, L 1, 2, 3, 4, and 5	.....	Smear and culture +	.....
17	5½	M	White and Filipino	Pain and swelling, right heel	2 wk.	Rt. os calcis	.....	Culture +	.....
18	13	M	Negro	Lump on head	.....	Occipital bone	.....	Guinea pig inoculation +	.....
19	1 10/12	M	Negro	Swelling over mandible	10 mo.	Rt. mandible	.....	Skin test and smear +	.....
20	5/12	M	White	Abscess of finger	2½ mo.	Rt. metacarpophalangeal joint 3d finger	.....	Smear +	.....
21	6/12	M	Negro	Cough; bumps on head; swollen areas on legs	1 mo.	Skull, rt. fibula, both ulnas, tibias, and femurs	Pulmonary involvement	Smear and culture +	Died of disseminated coccidioidomycosis

A report comprising a study of 26 cases of osseous coccidioidomycosis in children is presented. The data were obtained from the files of Kern General Hospital, Bakersfield, Calif. In all cases diagnosis was confirmed by skin tests, serological studies, smears, cultures, and/or autopsy findings.

Five cases were recognized during the period 1909 to 1930 and are summarized in Table 1. In these cases, four patients died of disseminated coccidioidomycosis; the fifth patient recovered, with complete healing of a metatarsal lesion. Because of lack of sufficient data, they are not considered further in the analysis of this series. The remaining 21 cases occurred during the period 1930 to 1950 and are presented in Table 2. Photographs and a summarized form of three of the cases are presented to better illustrate the disease.

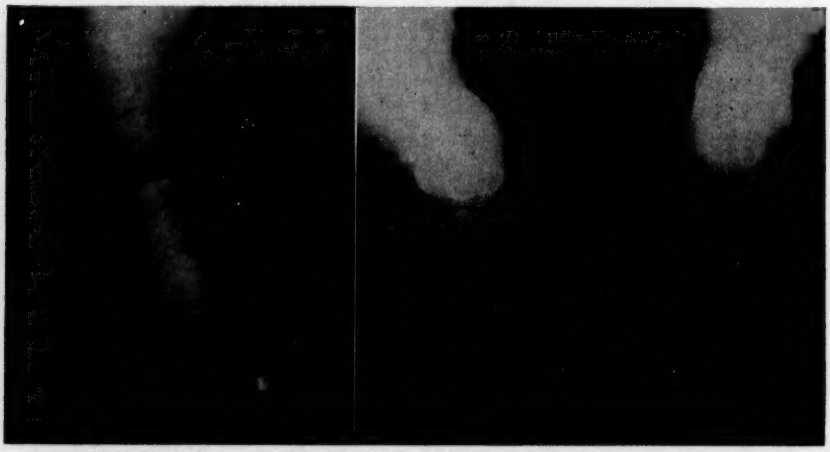

Fig. 1 (Case 1).—Soft tissue abscess and underlying osteolytic lesion of middle phalanx of right fourth toe.

REPORT OF CASES

Case 1.—S. G., a 5-month-old Mexican girl, had a single coccidioidal bone lesion (Fig. 1), which developed while pulmonary infiltration was present. There was complete healing of the bone lesion in 14 months.

The presenting complaints of this child were swelling and redness of the right fourth toe for two weeks and nonproductive cough and questionable fever for one week. Physical examination was completely unremarkable except for findings referable to the toe and right inguinal adenopathy consisting of one discrete node. Roentgenographic examinations revealed an osteolytic lesion of the middle phalanx of the right fourth toe and a minimal infiltration in the apex of the left lung field. The coccidioidal skin test was equivocal; the complement fixation test was reported as indicating rather severe coccidioidal infection.

The soft tissue lesion became fluctuant, and thin yellow pus was obtained which was positive on culture for Coccidioides immitis. During hospitalization the patient's course was characterized by a low-grade fever. After discharge the patient was followed in the outpatient clinic. The soft-tissue lesion healed slowly with subsidence of drainage and swelling, and the x-ray appearance of the bone lesion improved with deposition of calcium. The final chest x-ray revealed chronic changes in the inner zones of both lung fields. There was a positive complement fixation test throughout the entire period of observation.

CASE 2.—A. V., a 12-month-old Mexican boy, was seen with multiple coccidioidal bone lesions which had developed subsequent to pulmonary involvement; the osseous lesions healed over a three-year period. An accompanying decrease in titer of the complement fixation test occurred.

At the age of 7 months the patient was hospitalized for three days with the diagnosis of measles and bronchopneumonia. He was hospitalized again at the age of 10 months, because of an acute febrile illness. The chest x-ray at this time revealed some resolution of the parenchymal infiltration, as compared with the previous chest film. The coccidioidin skin test elicited a positive reaction, and a diagnosis of coccidioidomycosis of the lung was made. The patient became afebrile and was discharged after three days.

At 12 months of age the child was again seen in the outpatient clinic, with a febrile illness and generalized adenopathy. There were subcutaneous nodules on the right forehead and distal

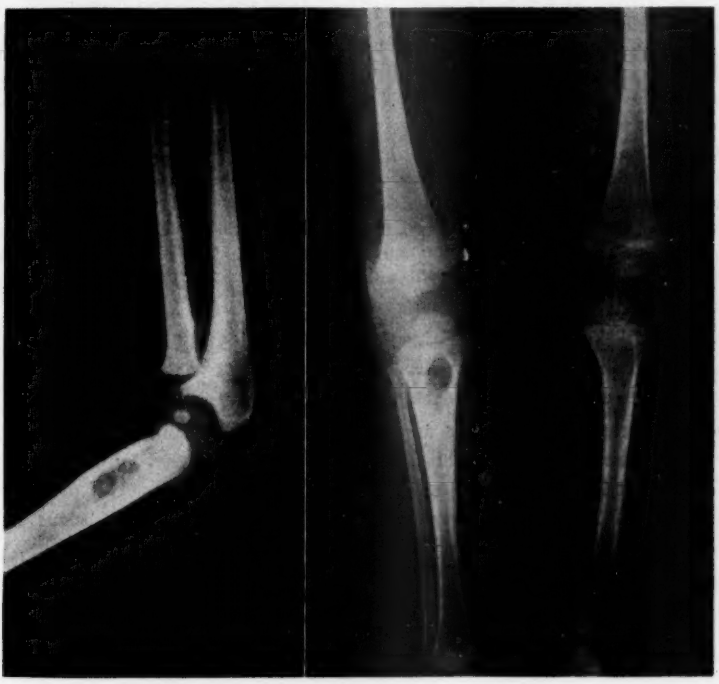

Fig. 2 (Case 2).—Unsuspected osteolytic lesions of tibia and humerous discovered during bone survey following diagnosis of coccidioidomycosis. There was no soft tissue involvement.

end of the right clavicle; x-rays revealed osteolytic lesions at these sites. The chest x-ray showed hilar adenopathy and hazy infiltrations of both lung fields. A subsequent bone survey disclosed unsuspected lesions of the first and fifth metacarpals, head of the right tibia, and lower end of the left humerus (Fig. 2). A repeat coccidioidin skin test reaction was 4+, and serological studies were interpreted as showing disseminated coccidioidomycosis.

At 14 months of age an osteolytic lesion of the left mandible was discovered, which was followed one month later by the development of a soft tissue abscess that drained. Smear and culture from the lesion were positive for C. immitis. At 21 months of age a decline in the titer of the serological tests was reported. Follow-up x-ray examination disclosed the lesions to be healed, with the exception of those of the right tibia and left mandible. Further serological studies at the age of 2 years 7 months revealed a continued gradual fall in titer.

At the age of 3 years 7 months, survey films showed a definite decrease in the size of the two remaining bone lesions. At this time the chest x-ray revealed an irregularity of the hilus, with a granular appearance of the inner and middle lung zones.

CASE 3.—D. S., a 6-year-old Negro boy, had multiple coccidioidal bone lesions and generalized adenopathy over a six-year period. There was no evidence of pulmonary involvement during the course of the osseous coccidioidomycosis.

The admission diagnosis in this case was "chronic lymphadenitis" due to coccidioidomycosis of the submandibular lymph nodes. A history of "tumors" since the age of 2½ years was obtained. Several of the masses had been removed from the right and left axillas and the left submandibular area. Subsequently to the excision at the latter site, drainage of creamy-white pus persisted for months.

Physical examination disclosed swelling of the left side of the nasal bridge and small, nontender, discrete nodes at the right and left angles of the mandible, with enlargement of the entire right side of the mandible. X-ray examination revealed osteolytic destruction of the left ramus and marked thickening of the right mandible. Roentgenographic examination of the chest was noncontributory. Smears from the biopsy specimens of the lymph nodes and left mandible contained coccidioidal spherules. The complement fixation test was positive; however, coccidioidal skin test reactions remained negative for two months after admission.

The patient was followed for three and one-half years and continued to have a persistent low-grade fever, despite the gradual healing of the lesion as shown clinically and by x-rays. Repeated serological tests revealed a persistently high titer. On the final visit the patient had been afebrile for one month. There was no clinical evidence of an active lesion. X-rays showed that the osteolytic areas had filled in, but the bony irregularity and enlargement of the right mandible remained.

COMMENT

One of us (J. W. B.) initiated this study when he found that he was seeing one or two patients with coccidioidomycosis of the bone every two to three months, either in private practice or at Kern General Hospital.

Roentgenograms demonstrated an osteolytic lesion, and it did not differ significantly from that seen in adults; this has been described in detail by Carter,[2] Miller and Birsner,[3] and others. There appeared to be no particular site of predilection, and any bone of the skeletal system might be involved. Multiple osseous lesions occurred in eight cases; three of these terminated fatally.

There apparently was no age of greatest incidence; four of the patients were under the age of 1 year. Coccidioidal pneumonia developed in one patient at the age of 2 weeks (born during a dust storm), and an osseous lesion appeared at the age of 1 month (Fig. 3). Males and females appeared equally susceptible, and, as has been previously reported for the disease in adults, osseous disseminated coccidioidomycosis occurred more frequently in Mexican and Negro persons; only two patients in this series were white.

The chief complaint of the majority of patients was painful, tender swelling; frequently a history of injury to the site was obtained. In only two cases was the presenting picture that of a severely ill child. In one case the lesion was an incidental finding discovered during an examination for another illness. In Case 7 additional unsuspected bone lesions were discovered after bone survey by x-ray.

2. Carter, R. A.: Infectious Granulomas of Bones and Joints, with Special Reference to Coccidioidal Granuloma, Radiology **23**:1-16 (July) 1934.

3. Miller, D., and Birsner, J. W.: Coccidioidal Granuloma of Bone, Am. J. of Roentgenol. **62**:229-236 (Aug.) 1949.

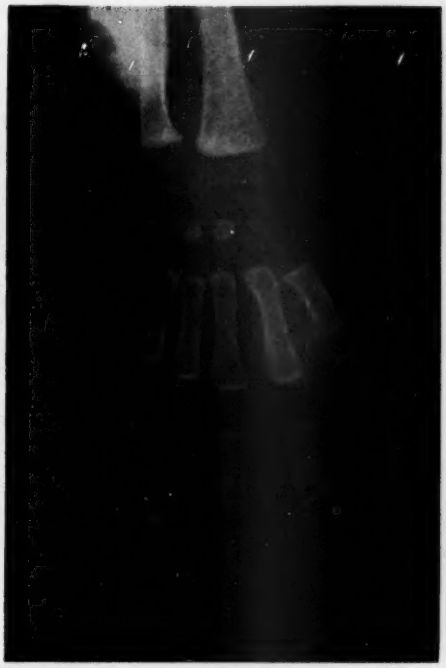

Fig. 3 (Case 4).—Osteolytic lesion of middle phalanx of right ring finger, which began at age of one month.

Fig. 4 (Case 5).—Soft tissue abscess accompanying osseous lesion of right maxillary bone and zygomatic process.

Fig. 5 (Case 6).—Coccidioidal lesions of frontal bone in 2½-year-old child.

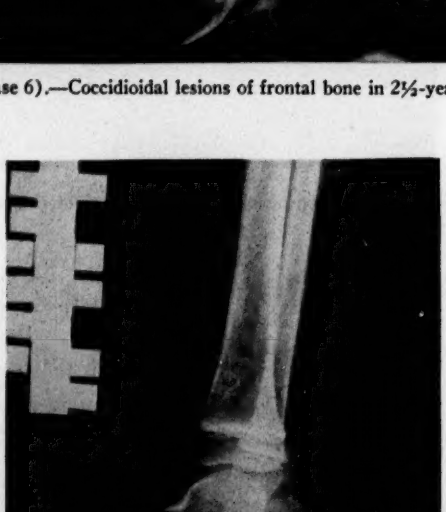

Fig. 6 (Case 7).—Involvement of left tibia and os calcis.

Five of the children had pulmonary lesions when admitted to the hospital. Two patients had pulmonary infiltration which cleared prior to development of the osseous lesions. In one patient a pulmonary infiltration (probably not coccidioidomycosis) occurred subsequent to bone involvement. Roentgenologic examination of the lung fields was noncontributory in 13 cases at the time of discovery of the osseous lesion.

Of the reactions in the 13 cases in which the results of skin tests were recorded, 10 were positive, 2 were equivocal, and 1 was negative. This is in contradistinction to the results seen in adults, in whom a negative skin test is not infrequent. When a child has an osseous lesion that does not respond to antibiotics, a skin test for coccidioidomycosis may be indicated, followed by the complement fixation test.

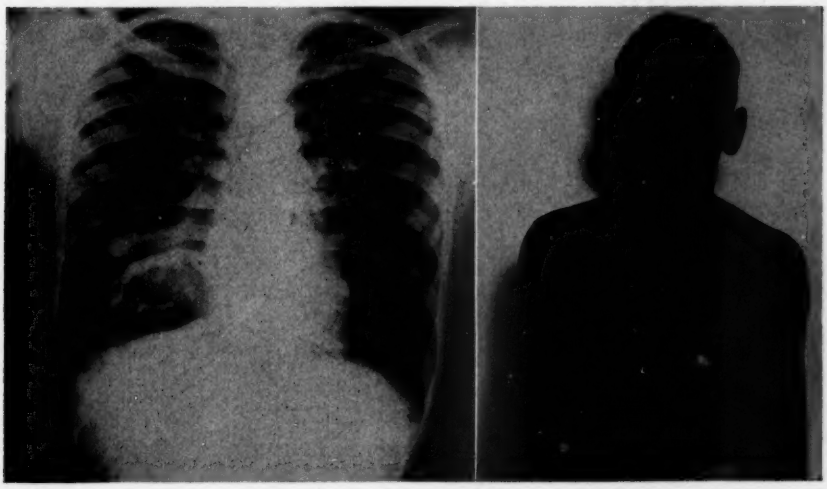

Fig. 7 (Case 8).—Soft tissue abscess and lesion of right 10th rib.

Contrary to the usual opinion, involvement of bone with coccidioidomycosis in children in itself does not connote a severe disease which ends fatally. In Cases 2 and 3 the patients had multiple osseous lesions and recovered (A. V. [Case 2] recently died of sympatheticoblastoma, and a very careful autopsy revealed no evidence of coccidioidomycosis). Noteworthy is the case of a patient, not included in this series, who, at the age of 12 years, had a clinical diagnosis of coccidioidomycosis of the ankle. After being asymptomatic for 18 years, including time that the patient served in the armed forces, the ankle became swollen and painful. X-rays at this time revealed two osteolytic lesions at the distal end of the tibia; a direct smear of a biopsy specimen disclosed C. immitis.

As seen from this series of cases, there may be involvement of the lungs as well as the skeletal system without rapid progression of the disease. The latter picture occurs only in those cases in which vital organs are involved with multiple lesions. This was seen in three of the cases in this series.

SUMMARY

Twenty-six cases of coccidioidomycosis of bone in children are presented.

The lesion is osteolytic in type, similar to that found in adults, and may occur as a single lesion or multiple lesions. It may or may not have an accompanying pulmonary lesion or cutaneous abscess.

As a rule, the process is chronic, with relatively good prognosis for eventual recovery.

Parenchymal infiltration of the lung frequently precedes the bone lesion, and at the time of osseous coccidioidomycosis there may be only residual pulmonary adenopathy.

In endemic areas the lesion is not uncommon, and elsewhere it may occur frequently enough that coccidioidomycosis should be considered when there are osteolytic lesions in children. An appropriate travel and previous residence history may suggest this diagnosis.

Dr. R. A. Carter, Chief of Radiology, Los Angeles General Hospital, and Dr. Joseph A. Weinberg, Chief of Surgery, Long Beach Veterans' Hospital, helped in the preparation of this paper.

Case Reports

HYDRANENCEPHALY

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HYDRANENCEPHALY is a congenital anomaly of the head that consists of complete or nearly complete absence of the cerebral hemispheres together with intact meninges and skull. It is associated with a normal-sized head when the condition is encountered early in infancy. Babies who have this anomaly present normal appearances and reactions during the first few weeks of life. Retardation of neuromuscular development and excessive enlargement of the head usually are not obvious until the child is several months old.

This anomaly was first described by Cruveilhier in 1835 and was named "l'anencephalie hydrocephalique" (hydrocephalic anencephaly).[1] This term later was consolidated into one word by Spielmeyer [2] in 1905, and hydranencephaly has been the accepted designation for this condition, although Yakovlev and Wadsworth [3] suggested the name "schizencephalies" after detailed histologic study of two cases. The latter authors considered the defect to be a true cerebral agenesia, with congenital clefts of the cerebral mantle and hydrocephalus. Other theories concerning etiology have been advanced but none have been proved. Edinger and Fischer [4] described a case in which the pia mater was present and contained an impression of the cerebral convolutions on its surface. Spielmeyer [2] described hydranencephaly in each of identical twins and concluded that it was caused by circulatory disturbances late in fetal life. In most cases, an intact meningeal circulation with normal pulsations has been observed during operation. Cohn and Neumann [5] considered severe bilateral porencephalia to be the proper classification for this condition.

Fellow in Pediatrics, Mayo Foundation (Dr. Olive).
From the Section of Pediatrics, Mayo Clinic (Dr. DuShane).

1. Cruveilhier, J.: Anatomie pathologique du corps humain, ou descriptions avec figures lithographiées et coloriées, des diverses altérations morbides dont le corps humain est susceptible, Paris, J. B. Baillière, 1829-1835, Vol. 1, p. 2.

2. Spielmeyer, W.: Ein hydranencephales Zwillingspaar, Arch. Psychiat. **39**:807-819, 1905.

3. Yakovlev, P. I., and Wadsworth, R. C.: Schizencephalies: Study of Congenital Clefts in Cerebral Mantle; I. Clefts with Fused Lips, J. Neuropath. & Exper. Neurol. **5**:116-130 (April) 1946; Schizencephalies: Study of Congenital Clefts in Cerebral Mantle; II. Clefts with Hydrocephalus and Lips Separated, ibid. **5**:169-206 (July) 1946.

4. Edinger, L., and Fischer, B.: Ein Mensch ohne Grosshirn, Arch. ges. Physiol. **152**:535-561 (July) 1913.

5. Cohn, R., and Neumann, M. A.: Porencephaly: Clinicopathologic Study, J. Neuropath. & Exper. Neurol. **5**:257-270 (July) 1946.

REPORT OF A CASE

A girl 4 weeks of age was admitted to the hospital because she had failed to gain weight and was restless. The baby had been born at full term after an uncomplicated pregnancy.

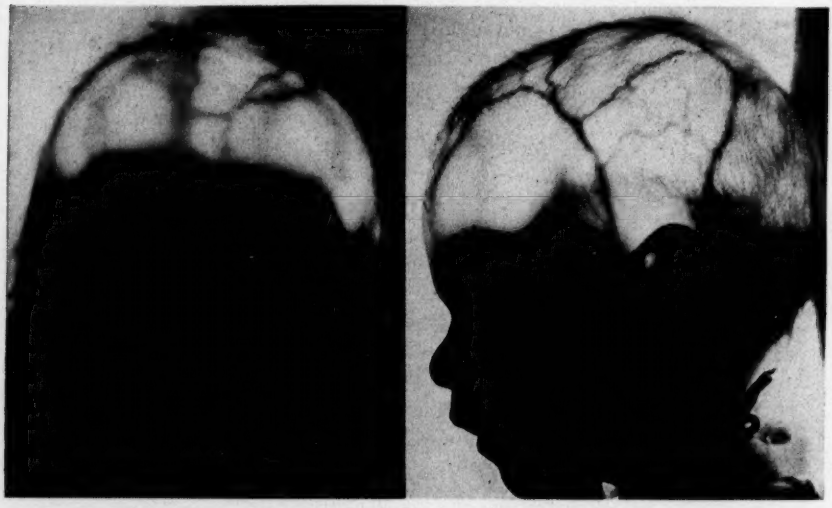

Fig. 1.—Photograph of the head, showing effects of transillumination. The source of light is behind the head.

Fig. 2.—Roentgenograms of the skull. Note incomplete ossification and separation of sutures.

She cried and breathed spontaneously; no cyanosis or pallor was observed. Weight at birth was 8 lb. 2 oz. (3.65 kg.). The mother had noted a high-pitched "whining" cry since the first few days of life, and this had persisted. The baby took feedings poorly, vomited frequently,

and failed to gain weight. She was restless and cried frequently. The family physician, shortly before referring the infant to the hospital, had noticed some enlargement of the head and failure to respond to bright light.

General examination at the hospital revealed a normal-appearing 4-week-old baby, who had a high-pitched cry and whose weight was 7 lb. 14 oz. (3.55 kg.). The movements of the head, trunk, and extremities were normal. She sucked vigorously and responded to noise, but not to light. Tendon reflexes were active, and the Moro embrace reflex was present. The ocular movements were dissociated. The cranial sutures were separated, and the cranial bones seemed to be floating. A tympanitic note was present when the skull was percussed. Transillumination of the head disclosed diffusion of light in all directions. The vascular shadows of the scalp were visible clearly, and several shadows at the base of the skull were observed (Fig. 1).

Roentgenographic examination of the skull showed incomplete ossification of the bones and separation of the sutures (Fig. 2). Electroencephalographic studies revealed no evidence of electric activity. The results of other laboratory studies were not unusual.

Because of the results obtained from transillumination of the head, a diagnosis of hydranencephaly was made and no further investigations were carried out. The infant was dismissed to her home, where she failed to thrive and gradually lost weight, finally dying at the age of 7 months. Necropsy was not done.

COMMENT

The condition encountered in this infant was a typical example of hydranencephaly, an anomaly that should be diagnosed during life. The importance of early diagnosis is obvious because the prognosis in such cases is hopeless and the parents of such a baby can be forewarned of the ultimate outcome. It is, however, difficult to diagnose early, since an infant with this disorder usually appears normal at birth, and for several weeks thereafter no obvious symptoms occur that prompt the parents to seek medical opinion. Enlargement of the head may not be noted for several months. Evidence of retarded development may also be delayed since the newborn baby apparently utilizes lower centers of the brain in early life, and mothers expect little in the way of responsiveness except for sucking and crying.

Transillumination of the head is the most valuable single aid in diagnosis. This is a simple maneuver that can be performed with a minimum of equipment. A bright light is held behind the head of the infant in a darkened room. This is much the same as the technic that is employed for the demonstration of a hydrocele. It is an impressive sight to note the entire head glowing with a red light, while the vessels of the scalp, the venous sinuses, and the basal structures of the brain stand out in dark contrast to the lantern-like glow of light through the head. When the light is placed at the occiput, and the eyelids are held open, pinpoints of light can be seen at the pupils. Strasburger [6] described the phenomenon of transillumination in relation to hydrocephalus in 1910, when he observed that light could be seen through the head of a child if the cerebral tissue was not greater than 1 cm. in thickness. More recently, Hamby, Kraus and Beswick [7] reported a series of seven cases of hydranencephaly in which transillumination of light through the head was a constant feature. They emphasized also that this diagnostic test removes hydranencephaly from the status of a curiosity observed at necropsy and permits the proper diagnosis to be made during life. The importance of this statement is attested by

6. Strasburger: Transparenz des Kopfes bei Hydrocephalus, Deutsche med. Wchnschr. **1**:294 (Feb. 10) 1910.

7. Hamby, W. B.; Kraus, R. F., and Beswick, W. F.: Hydranencephaly: Clinical Diagnosis; Presentation of 7 Cases, Pediatrics **6**:371-383 (Sept.) 1950.

the report of a case that involved a one-week-old baby who was pronounced normal by a competent physician and offered subsequently for adoption.[8]

Electroencephalographic tracings demonstrate usually complete absence of electric activity in all leads, as occurred in our case. A small area of electric activity may be discovered in one part of the head, as was evident in one of Beswick's [8] cases. This was proved to be produced by a remnant of cerebral tissue.

Careful examination of the eyes also may be helpful in establishing the proper diagnosis. While the pupils may react to light, no evidence indicates that the infant has a true appreciation of the presence of light or shadow. Ocular movements have been described as wandering and uncoordinated.

The ultimate prognosis for patients with hydranencephaly is poor, although Edinger and Fischer [4] described one child who lived for three and one-half years. In most cases that have been reported, death has occurred before the age of one year.

8. Beswick, W. F.: Schizencephalies with Hydrocephalus: Clinical Diagnosis of Severe Cerebral Agenesis, New York J. Med. **48**:2364-2366 (Nov. 1) 1948.

PERFORATION OF THE STOMACH IN THE NEWBORN

Report of Two Cases with Necropsy Findings

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PERFORATION of the stomach in the newborn infant is rare. It is believed that the cases here reported constitute the 21st and 22d instances of such perforation. One of them is the second case of pyloric atresia complicated by perforation.

In a comprehensive review of the literature in 1941, Bird, Limper, and Mayer [1] found 245 recorded cases of peptic ulcer in children 15 years of age and under. Of these, 42 were in infants 0 to 14 days of age, with a ratio of duodenal to gastric ulcers of about two to one. It was pointed out that in such ulcers recognized in the early days or months of life, serious bleeding or perforation or both are characteristic. During later childhood, however, the incidence of pyloric stenosis and perforation increases, with a decrease in the incidence of serious hemorrhage.

REPORT OF CASES

CASE 1.—T. W., a 3-day-old white boy baby, was brought to the outpatient department of Children's Mercy Hospital on Nov. 9, 1950. He was apparently normal until approximately 18 hours before admission. At this time it was noted that the infant was slightly jaundiced, and the abdomen was distended. The child became progressively more toxic and lethargic and was in apparent pain. The past history revealed that the infant had been given formula feedings every four hours without apparent difficulty. There was no vomiting, and bowel movements and urination were normal. There was no history of gastric gavage or trauma. The family history revealed that the mother, sextigravida and quintipara, after an uneventful period of pregnancy, had a normal spontaneous delivery with the labor lasting six hours. The mother and father were Rh positive.

Physical examination revealed a white boy baby, slightly jaundiced, who appeared acutely ill and apparently in severe pain. The pulse was fast and weak. The temperature was 100 F. (rectal). The abdomen was markedly distended. While rigidity was not present, the abdomen seemed "firm." It was tympanitic throughout. Bowel sounds were absent.

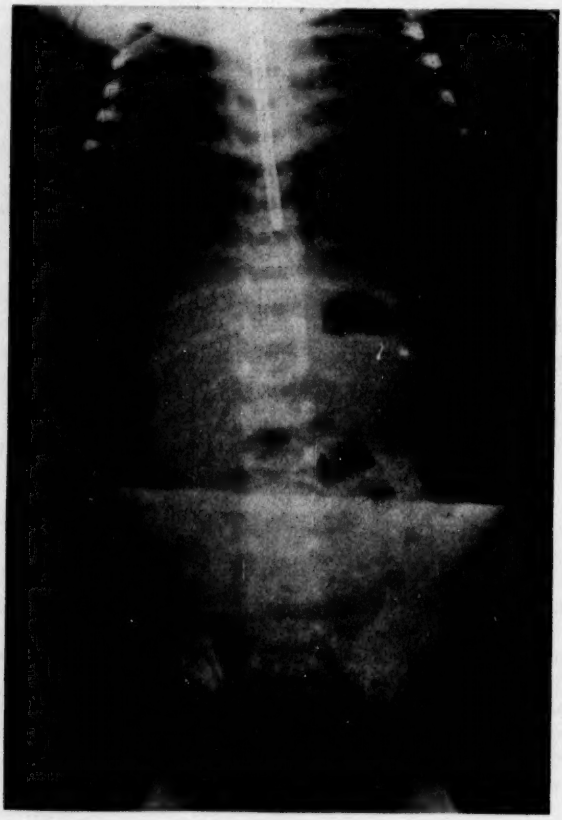
Roentgenograms of the abdomen with the patient in the recumbent and upright position showed a large amount of free gas and free fluid in the peritoneal cavity (Figure). The viscera were revealed as a conglomerate mass in the midabdomen. The diaphragm appeared smooth and regular. The findings were those of a pneumoperitoneum and a hydroperitoneum.

The laboratory findings were as follows: hemoglobin 17 gm. per 100 cc., white blood cell count 5,100, red blood cell count 5,750,000, neutrophiles 49, lymphocytes 44, monocytes 7, icteric index 30 units, Kahn and Kline tests negative, and Rh positive. Gastric contents were positive for bile and negative for trypsin activity.

From the Children's Mercy Hospital, Kansas City.

1. Bird, C. E.; Limper, M. A., and Mayer, J. M.: Ann. Surg. **114**:526-542, 1941.

After the intravenous administration of fluids, including blood, the patient was prepared for surgery. Under open-drop ether anesthesia, a midline incision was made. When the peritoneum was incised a large amount of free gas escaped together with bile colored fluid. Upon exploration of the abdomen a 2 cm. tear along the greater curvature of the stomach was found, with gross spilling of the contents into the abdominal cavity. The wall of the laceration or tear was excised and the defect closed with a running suture of fine chromic absorbable surgical suture, reinforced by interrupted sutures through the serosa. No other areas of perforation were found.



Roentgenogram from Case 1, taken in the upright position. Pneumoperitoneum and well-defined fluid level are apparent.

The postoperative course was progressively downhill, although constant gastric suction, antibiotics, and fluids by vein were given. The patient died 18 hours later. At autopsy the body was that of a fairly well-developed white baby boy. There was a recent midline incision closed with silk. The entire skin was quite cyanotic. When the peritoneal cavity was opened, it was found that the entire cavity was covered by a fibrinous exudate, with some yellow-gray material resembling milk or curds in each lateral gutter. The peritoneal surface of the bowel varied from a gray to reddish-gray color. The greater curvature of the stomach showed a sutured defect measuring 3 cm. in length. The abdominal organs were in the normal position with no areas of atresia or stenosis. The biliary tract was patent throughout. The remainder of the examination showed no significant abnormalities.

Section through the stomach, from the material removed at the time of operation, showed marked hemorrhage of the mucous membrane with a remarkable degree of edema of the submucosa. The muscularis showed infiltration between the muscle bundles by leucocytes, polymorphonuclears, monocytes, and plasma cells. On the serosal surface there was also extensive hemorrhage, and an acute fibrinopurulent exudation was seen near the point of perforation. In this area of perforation there was a necrotic ulcer containing numerous pus cells, red cells, and necrotic fibrin.

Section taken from the gastric wall adjacent to the repaired perforation in the autopsy specimen showed a fairly intact mucosa. The walls of the stomach had undergone autolytic necrosis about the suture material. The muscularis seemed to be thin here. The serosa was covered with necrobiotic exudate containing large masses of bacteria. There does not, however, appear to be any congenital defect in the muscularis, since both inner circular and outer longitudinal muscle bundles were intact except in the area of closure.

CASE 2.—C. was a 13-day-old white boy. The mother of this baby was a 21-year-old primigravida and nullipara, who had had a normal period of pregnancy. After a labor of 11 hours 8 minutes, with 100 mg. of meperidine (demerol®) hydrochloride and scopolamine hydrobromide (1:200) being used as a sedative, the baby was born spontaneously. At birth the infant weighed 6 lb. 1 oz. (2750 gm.) and measured 19 in. (48 cm.) in length. The facial features were mongoloid. Shortly after birth the infant became cyanotic and was promptly examined by laryngoscope; a mucous plug was removed from the trachea, with prompt improvement in the color. The baby was put into an incubator, and oxygen, antibiotics, and 2 minims (0.123 cc.) of nikethamide (coramine®) as a respiratory stimulant, were given. Later, the administration of oxygen was discontinued, and a formula feeding was started.

On the third day after delivery the baby began to have fever with an occasional regurgitation of the formula. The temperature fluctuated from 99 F. to 102 F. (rectal). The bowel movements consisted of meconium. Hypodermoclysis of Hartman's solution was given. Vomiting became more frequent and the abdomen distended. The patient became progressively worse with markedly labored respirations and increased cyanosis and vomiting, and died on the 13th day after birth.

When the peritoneal cavity was opened at autopsy it was found to be distended by air and contained a large amount of purulent exudate. The stomach was enormously distended, and the bowel was surrounded by fibrin. When the left lobe of the liver was lifted, a perforation on the anterior surface of the stomach, which measured 2 mm. in its greatest diameter, was found. The first portion of the duodenum was distended. There was a communication between the first and second portions of the duodenum by a very narrow channel. The first portion was filled with a yellowish mucoid material and the second portion with a dark green liquid. Histologic examination of the lungs showed scattered areas of atelectasis and hemorrhage into the alveoli with focal emphysematous zones and patchy bronchopneumonic consolidation. Section through the stomach near the area of perforation showed increased mucus secretion. The submucosa was hyperemic. The muscularis mucosa was intact. The inner circular and outer longitudinal layers of muscle were intact. There was a thick fibrinopurulent exudate on the surface of the stomach, and in some areas there was considerable edema and some leucocytic infiltration of the subserosa. The peripancreatic nodes showed an acute hyperplastic lymphadenitis.

The diagnosis was a congenital stenosing lesion of the duodenum with gastric perforation and generalized peritonitis, and bronchopneumonia with focal pulmonary atelectasis.

COMMENT

As pointed out by Herbut,[2] in 14 of 15 reported cases of rupture of the stomach in the newborn, the perforation was secondary to gastric ulcer. In the remaining case, only hemorrhage in the borders of the perforated area was observed. In reporting the 16th case, however, Herbut seems to have demonstrated a congenital defect in the musculature of the stomach wall with rupture apparently occurring as a result. Burnett and Halpert,[3] recorded the 17th case of perforation and the 1st

2. Herbut, P. A.: Arch. Path. **36**:91-94, 1943.
3. Burnett, H. A., and Halpert, B.: Arch. Path. **44**:318-320, 1947.

with atresia of the pyloris. They noted that the muscle layers of the stomach, while broad in the uninvolved portion, were thinned out near the site of perforation. Potter [4] described a case in which the muscle layers became gradually thinner and disappeared at the margin of the tear in the stomach wall.

Various explanations have been advanced as to the cause of gastric and duodenal ulcers in the newborn. Ladd and Gross [5] have described them in stillborn infants. Guthrie [6] believes that they arise after birth as a result of circulatory instability, as observed in prolonged labor with devitalization of the mucosa and increased susceptibility to digestion by gastric juice. Embolism, thrombosis, vascular disease, direct injury to the mucosa by gavage, and vomiting have all been suggested as causative factors. In 39 cases of rupture of the intestine in the newborn, collected by Russell,[7] 28 were thought to have become perforated during birth. It was his opinion that the pressure of the birth canal on an intestine filled with meconium produced the rupture. In 13 of the 39 cases, rupture of the intestine occurred proximal to an obstruction. In so-called spontaneous rupture of the stomach in adults Wolf [8] states:

> The force acts from within the viscus and distention of the stomach by food either paralyzes the nervous mechanism, causing further dilatation, fermentation, and eventually rupture, or interferes with the circulation, producing softening of the wall and subsequent perforation.

It would seem, then, that external pressure of the birth canal, distention of the stomach with or without obstruction, trauma of the mucosa (as by gavage tube) with infection, digestion and perforation, and congenital absence or thinning of the muscle play a part. All of these factors, however, do not seem to apply in all cases, since absence or thinning of the muscle layers has been observed in only three cases.

Diagnosis of the above condition is difficult. The usual history is that of a normal-appearing infant following an uneventful delivery. Within the first few days after birth the infant refuses feedings and becomes apathetic. This usually, but not always (Case 1), is followed by vomiting, abdominal distention, and toxemia. Though the abdomen is distended, muscle rigidity as seen in older patients may be absent. Once rupture of a viscus is suspected, and this possibility must not be overlooked merely because of the patient's age, a roentgenogram in the upright position will show free air in the peritoneal cavity.

The treatment is obviously surgical. So far as can be ascertained, closure of a perforated stomach in a newborn infant has been successfully accomplished in only one instance. Leger, Ricard, Leonard and Piettte [9] reported the closure of a

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4. Potter, E. L.: Pathology of the Fetus and the Newborn, Chicago, Year Book Publishers, Inc., 1952, pp. 295-296.

5. Ladd, W. E., and Gross, R. E.: Abdominal Surgery of Infancy and Childhood, Philadelphia, W. B. Saunders Company, 1941, pp. 19-22.

6. Guthrie, K. J.: Arch. Dis. Childhood **17**:82-94, 1942.

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perforation of the stomach in a 6-day-old infant with survival. The closure was done 16 hours after the onset of symptoms. However, the condition should not be as hopeless as this record would indicate. In the few cases in which surgical closure has been attempted, considerable time has elapsed between the time of perforation and the operative closure. Early diagnosis and immediate resort to surgery should offer a more hopeful outlook in these cases.

SUMMARY

The 21st and 22d cases of perforation of the stomach in the newborn infant are reported. In one of them, surgical closure of the perforation was done. Necropsy findings in both are recorded. Perforation in one instance was secondary to an ulcer, and in the other to atresia of the duodenum. The etiology, diagnosis, and treatment are briefly discussed. While successful surgical closure has been accomplished in only one instance, early diagnosis and prompt operation may offer some hope in these cases.

F. C. Helwig, M.D. supplied necropsy descriptions and other assistance, and Raymond Anderson, M.D., gave us permission to include Case 2 in this report.

Society Transactions

CHICAGO PEDIATRIC SOCIETY

Craig D. Butler, M.D., President
Regular Meeting, Nov. 20, 1951

Further Studies of the Influence of Histamine on Qualitative Platelet Activity, with Especial Reference to Its Action on the Blood of the Hemophiliac. Dr. Heyworth N. Sanford, Dr. F. Richard Hall (by invitation), and Dr. Stuyvesant Butler (by invitation).

It was found by clinical and experimental studies that histamine when given intravenously or subcutaneously directly increased the qualitative disintegrative ability of the blood platelets. It has no effect on the total platelet count (qualitative action) until shock-like levels are obtained. It then causes a decrease in the number of the circulating platelets in the blood.

The qualitative action may be in part due to an antagonistic action between heparin and histamine. Heparin in high concentrations does not inhibit completely the increased tendency toward in vitro agglutination of the platelets exposed in vivo to histamine.

In the administration of the histamine to hemophiliac patients, the following plan has been followed: 0.1 mg. of histamine is given the first day, 0.2 mg. the second, 0.4 mg. the third, 0.8 mg. the fourth, and 1.0 mg. the fifth day. If a longer series is desired, the amount of histamine is not increased above 1.0 mg. By intravenous injection the histamine is given in 250 cc. of isotonic sodium chloride solution at a rate of 20 drops per minute. By hypodermoclysis the same quantity is given at the rate of 10 drops per minute.

In 19 out of 22 hemophiliac boys, the procedure decreased the coagulation time of the blood by increasing the platelet disintegration. It was a complete failure in three of the boys. This action is not permanent and tends to become less efficient as more injections are given. However, it is sufficient to cause cessation of bleeding and to decrease the coagulation time of the blood to its normal value for that person in the majority of instances.

Practical Points in the Diagnosis and Treatment of Congenital Malformations of the Heart. Dr. Benjamin M. Gasul, and Dr. Egbert H. Fell (by invitation).

Craig D. Butler, M.D., President
Regular Meeting, Dec. 18, 1951

Symposium on Primary Tuberculosis in Children: The Primary Infection. Dr. E. T. McEnery.

The subject matter for discussion in this symposium may seem very elementary and didactic, but the problem of tuberculosis is a very timely subject. The picture of tuberculosis has been changing, especially during the past five years. Much has been written, and many new drugs have been used in treatment of this disease. The reports on the use of these drugs have been extensive, and now, after a period of five years, we feel that the subject should be brought up to date.

Tuberculosis is usually caused by direct infection from a person with active tuberculosis, and, in the great majority of cases, the bacilli are conveyed by droplet infection through a cough or sneeze and are aspirated by the recipient. The first infection is usually just beneath the pleura, and the bacilli soon create a small area of tuberculosis known as the primary focus. Almost as soon as this inflammatory process takes place, bacilli begin to leave the primary focus and are carried through the lymphatic vessels to the nearest group of lymph nodes draining the area in which the primary focus lies. This combination of the primary focus with a corresponding lesion in the regional lymph nodes and the interfocal zone between the two is known as the primary complex.

The reaction around the primary lesion may vary greatly. The size and intensity of the reaction depend upon the extent of the tuberculous disease at the port of entry and also upon the age and race of the patient. The usual reaction is of a benign nature, but sometimes the lesion may caseate, become excavated, and progress to bronchogenic spread. Unfortunately there is no way of telling when one of the benign lesions may progress to a serious outcome. It is because of the unknown factor that all patients should be watched closely.

The early primary infection is devoid of symptoms and physical findings. The indication is usually picked up on a routine survey by a positive tuberculin test. This means that the patient had a tuberculous infection. The next step is to find the contact and remove the child from the source.

Active treatment at this stage is not necessary. The child should have rest and good food and should be under close medical supervision. A check by x-ray should be made every three months, especially if the child is under 1 year of age. The use of streptomycin does not preclude or avoid the onset of complications. Many resistant strains of bacilli have been found in early cases, and meningitis has developed while the drug was being given.

Criteria for Sanatorium Care of Children with Tuberculosis. Dr. M. R. Lichtenstein.

This paper was published in full in the March, 1952, issue of the American Journal of Diseases of Children, page 292.

Bronchial Complications of the Primary Lesion. Dr. Benjamin M. Levin.

DISCUSSION

Dr. Edward A. Piszczek: There is little I can add to the scholarly discussions that have been given except to say that the pediatricians are still busy taking care of the primary problems of tuberculosis, as evidenced by the open cases that we have. Our death rate in Chicago is four times as high as that of New York City. We still have a little problem in the suburbs, but are attacking it more vigorously because we do not have a shortage of sanitarium beds. We hospitalize all the cases in the suburbs immediately. Most of our child problem is tuberculous meningitis. We have been supplying patch test materials to the Health Department and infant welfare clinics and will be glad to do the same for any pediatrician who practices in the suburbs. We will also furnish x-rays free for any children referred by a private physician at a suburban hospital, the West Side office, or at the mobile unit that visits the towns in the suburban areas monthly.

I agree with Dr. Lichtenstein that we have too few criteria for the admission of children. We do have many children who have primary tuberculosis because of contact with tuberculous relatives. Although some of these children get well in spite of the environment, others break down into reinfection tuberculosis. We have an interesting example in a child of 3, who had a primary tuberculosis a year ago. The disease had quieted down for six months and now reinfection tuberculosis with cavitation has developed. The problem was discussed at a staff meeting, and the films were reviewed by Dr. Jerome Head and others. It was decided to send her home with a cast on, and we will watch her and see what happens. Most of these children will get well with plenty of rest, as Dr. Lichtenstein said, and why certain patients have reinfection at such an early stage is a question.

Dr. Eugene T. McEnery: I cannot agree with putting a body cast on a child with the idea of rest. I think that is carrying the idea of rest too far. If the child is seen early enough, you do not have to limit activities. I will be much interested in knowing how this child progresses. If a child is sick enough to be in bed, he will lie down and rest. We have all seen children in body casts, and, if it is a young child, one can realize what a predicament that child will be in.

Dr. Karl H. Pfeutze: I should like to ask Dr. Piszczek why chemotherapy was not used in that case. Obviously a 3-year-old child in whom a primary infection develops does not have her share of resistance, and I should think three or four months of chemotherapy would be in order. Certainly one need not worry about resistant organisms in four months.

Dr. Edward A. Piszczek: That child had six months of chemotherapy at Cook County Hospital, was symptomless, and is reinfected. The reason for putting on a cast was that the

cavity was in the superior portion of the right lower lobe. Pneumothorax or pneumoperitoneum cannot be used in a child. The easiest thing would be surgery, taking out the lower lobe. The x-rays have been seen by a number of good men. The child's father is a veteran and had an infection himself. The mother does not know how to handle the child properly, and the only way she could be put at rest was by a body cast. The child has had a series of streptomycin and *p*-aminosalicylic acid and is being given *p*-aminosalicylic acid again three times weekly.

DR. M. R. LICHTENSTEIN: The question was asked whether it is necessary for a child to have a positive sputum to be admitted to Municipal Tuberculosis Sanitarium. It is not necessary if there is evidence of dissemination which is probably tuberculous. Such children require immediate treatment. We do not expect absolute proof that the patient has tuberculosis in every instance, in such circumstances. However, if the child is not very ill and does not require immediate treatment, we prefer to wait for proof that the child has tuberculosis. In cases of pulmonary tuberculosis where there is time to investigate, we wish to have positive evidence by gastric lavage culture, guinea pig inoculation, or bronchoscopic aspirate culture. We do not always demand this if there is evidence that the diagnosis of tuberculosis is highly probable. Such evidence is recent tuberculin conversion or prolonged contact with open tuberculosis.

Craig D. Butler, M.D., *President*
Regular Meeting, Jan. 15, 1952

Subdural Effusion Following Purulent Meningitis. DR. ABRAHAM LEVINSON.

T. H., a 4-month-old Negro girl, was admitted to the Cook County Children's Hospital with a history of fever and vomiting for one day. She had had an upper respiratory infection 10 days prior to admission, but had not had fever until one day before admission. The past history was essentially unremarkable. On admission the child appeared acutely ill, assumed a position of opisthotonos, and had a bulging fontanelle. Divergent strabismus was noted. The pupils were small, round, and equal, and reacted poorly to light. There were nuchal rigidity and positive Brudzinski and Kernig signs. The skin was clear; no rash or petechiae were noted. The temperature was 104.4 F. (rectal). The remainder of the physical examination showed no significant abnormalities.

Spinal puncture on admission yielded a cloudy fluid with a cell count of 40 per cubic millimeter, 10 lymphocytes and 30 polymorphonuclear leucocytes. The Pandy reaction was 4+; protein 160 mg. per 100 cc.; glucose 26 mg. per 100 cc. The smear showed Gram-positive diplococci. A diagnosis of pneumococcic meningitis was made, and the child was given 300,000 units of procaine penicillin intramuscularly and 2 grains (0.13 gm.) of sulfadiazine per pound (0.5 kg.) of body weight per 24 hours intravenously in divided doses at six-hour intervals. The sulfonamide was discontinued the next morning when the diagnosis was substantiated.

On the morning after admission the child had a generalized convulsion, which was controlled with barbiturates. The following day she was afebrile but had a lateral nystagmus, twitching, and a bulging fontanelle. She remained rigid and semicomatose until four days after admission, when she began to take oral feedings. The general condition, however, remained unchanged.

A repeat spinal tap on the fifth day after admission showed a clear fluid, negative Pandy reaction, glucose 67 mg. per 100 cc., and protein 22 mg. per 100 cc. There were no cells. Because of the poor course and full fontanelle, a subdural tap was done on the following day and 25 cc. of xanthochromic fluid was removed from each side. This was repeated four days later, at which time 20 cc. of fluid was found on the right side and 15 cc. on the left. Two days later burr holes were made by the neurosurgeon, and subdural hematomas with membranes were found bilaterally.

The second patient, G. W., an 11-month-old Negro boy, was admitted to the Cook County Children's Hospital on Oct. 9, 1951, with a history of fever for about three days and a bulging fontanelle for about two days prior to admission. The baby was extremely irritable, refused feedings, and vomited. The remainder of the history showed nothing significant.

On physical examination the baby appeared acutely ill and extremely irritable. The temperature was 102.4 F. (rectal). The fontanelle was tense and bulging. The pupils were

round and equal and reacted to light. There were nuchal rigidity and a suggestive Brudzinski sign. Reflexes were active. There were no other significant findings.

Spinal tap on admission yielded turbid fluid with a cell count of 865, 98% segmented cells and 2% lymphocytes. The Pandy reaction was 3+; protein 36 mg. per 100 cc.; glucose 55 mg. per 100 cc. The smear showed Gram-negative pleomorphic rods. A diagnosis of Hemophilus influenzae meningitis was made, and the infant was given aureomycin hydrochloride, 200 mg. as the first dose and 100 mg. every six hours thereafter, by intravenous administration.

After two days the child was alert and took feedings, but had occasional episodes of vomiting. The fontanelle remained tense. A low grade fever, 100 to 101.6 F. (rectal), persisted. The child was maintained on orally administered aureomycin hydrochloride.

A repeat tap on Oct. 16, 1951, yielded clear fluid and no cells. The Pandy reaction was negative; glucose 55 mg. per 100 cc.; protein 16 mg. per 100 cc. The culture was negative for pathogens.

Because of the increase in head size and the large anterior fontanelle, a subdural tap was done 10 days after admission for the possibility of subdural hematoma. From the right side 45 cc. of xanthochromic fluid was removed and from the left side 50 cc. On the repeat subdural tap on Oct. 23, 40 cc. of fluid was removed from each side. On the same date, a spinal tap showed clear fluid with a cell count of 12, 42% polymorphonuclear leucocytes and 58% lymphocytes. The Pandy reaction was negative; protein 18 mg. per 100 cc., glucose 62 mg. per 100 cc.

On Oct. 26, 1951, 55 cc. of xanthochromic fluid was removed from the right side and 45 cc. from the left. On Oct. 29 the neurosurgeon made bilateral burr holes and found bilateral subdural hematomas with membranes. The hematomas were drained. The postoperative course was good; there were no signs of paralysis. On Nov. 12 a craniotomy was done, a left-sided bone flap turned, and the membrane removed from that side. The right side was done one month later.

At present the child is asymptomatic, sits and stands, feeds well, and is alert and active. There are no signs of paralysis, the anterior fontanelle is depressed, and the head is not enlarged. The child was discharged in excellent condition.

These two children had meningitis, one pneumococcic, the other influenzal. It was decided to do a subdural tap in each case because the patient was not doing well.

I do not believe subdural effusion follows meningitis frequently. However, the condition should be kept in mind when a child who has meningitis and has been given antibiotics does not recover within a reasonable time. It may be necessary to differentiate between subdural effusion and encephalitis as the cause for prolongation of the illness. The final diagnosis is made by subdural tap. In infants, before the anterior fontanelle is closed, a 22-gauge needle is inserted into the lateral angle of the fontanelle immediately below the skull and directed horizontally. If no fluid is obtained, the needle is directed upward and downward. Under normal conditions, no blood or fluid is obtained. In subdural effusion, from 5 to 100 cc. of xanthochromic fluid is obtained. The chemistry of the fluid is nearly that of blood. We usually culture the fluid, and, as a rule, it is negative. In some of our cases, however, it was positive.

The pathogenesis of subdural effusion is still uncertain. It seems plausible that it is analogous to pleural effusion. However, why is it that in empyema in infants there is purulent fluid, and in subdural hematoma the subdural fluid is xanthochromic? How do the membranes occur? It is also strange that instead of accumulating in the subarachnoid space, fluid occurs in the subdural space. Further study may answer these questions.

As to treatment, most neurosurgeons believe that when the diagnosis of subdural effusion or hematoma is made, the child should be operated on immediately. I believe it better to wait for 6, 7, or 10 days, at which time the membrane is usually formed.

Should every patient with meningitis have a subdural tap? My answer is no; just as not every patient with poliomyelitis or tetanus needs a tracheotomy. The only candidates would be those who are not doing well in spite of antibiotics after seven or eight days and those who have a bulging fontanelle. I do not believe subdural taps are dangerous or difficult; certainly they are much easier to perform than a cisternal puncture or spinal tap. However, one should be cautious because there is the remote possibility of infecting the subdural space.

Hypotonia and Its Treatment with Strychnine. Dr. Meyer A. Perlstein and Dr. Irving D. Mizell.

Most children with neuromuscular handicaps due to involvement of the brain have motor syndromes which are characterized by spasticity, rigidity, or other forms of hypertonia. This has resulted in a search for drugs to release muscle spasm. From a clinical viewpoint, however, patients with hypotonia are much more difficult to treat than those with hypertonia. This is particularly true in children who have cerebral palsy characterized by hypotonia, in whom the muscles are not strong enough to carry braces and in whom the tone is so decreased that the children may actually seem to have muscular dystrophy.

This preliminary report is to indicate our experience with the use of strychnine in the management of the hypotonic state. Four children are used to illustrate various types of hypotonia. Three of these children have been given strychnine with variable results, and one has not yet been treated.

Case 1.—This 12-year-old boy has a classical syndrome of pseudohypertrophic muscular dystrophy. He is in a wheel chair and is unable to lift his arms above his head. We have been unable to increase any of the muscle tone in this child by the use of strychnine.

Case 2.—This 7-year-old girl has muscular dystrophy of the scapulohumeral type. She is able to walk with marked lordosis; the greatest involvement is in the upper extremities. On therapy with strychnine there has been some improvement in muscle tone. Since spontaneous remissions are seen in this type of case, and since the mother was not able to differentiate between the reaction to the placebos and the reaction to the medication given, it is questionable whether the improvement may not be temporary and adventitious. We know that every time she has an upper respiratory infection she has a relapse, and during convalescence she improves.

Case 3.—This patient is a 4-year-old girl with Oppenheim's disease. This type of muscular dystrophy can generally be recognized at birth or very shortly thereafter. This child was admitted today and has not yet been put on a strychnine regimen. She is mentioned primarily to illustrate another form of hypotonia.

Case 4.—The final patient has cerebral palsy of the atonic athetoid type. The atonia in this child was so great that no spontaneous athetoid movements were visible. When originally seen, the symptoms of the child resembled those of Oppenheim's disease clinically. However, differentiation could be made in that it was possible for him to develop tension in his muscles on effort. After strychnine treatment was started, there was marked increase in tone so that he not only has developed spontaneous movement of the trunk and lower extremities, but is also able to maintain his head in an erect position. He is also able to sit in a chair without flopping over. Knee jerks, which were not present before administration of strychnine, are easily obtained, and, when lifted by hands passed under the axillae, he no longer "falls through" the lifter's hands.

Nux vomica tincture was originally used, so that we might gradually increase and decrease dosages. A dropper was used which delivered approximately 40 drops per cubic centimeter. The initial dosage was 15 drops three times a day, gradually increased until a pharmacologic effect was obtained. The first symptoms noted were diarrhea and vomiting. In the last patient with cerebral palsy, the dose was increased to 55 drops, or the equivalent of approximately 1/50 grain (1.2 mg.) of strychnine three times a day. On this dose the child had one convulsion, after which the dose was reduced to 45 drops three times a day. In all cases, placebos were also employed in order to eliminate any possible psychologic effect.

At present an aqueous solution of strychnine is being used instead of nux vomica tincture in order to eliminate the variability in strychnine content of various nux vomica mixtures, and also to eliminate the variability in the number of drops delivered per cubic centimeter with various-size droppers. With the aqueous mixture, 16 drops are uniformly delivered by the standard dropper per cubic centimeter of solution.

No good clinical method for determination of muscle tone exists. The physiologic method for determining muscle tone involves placing a needle into the muscle. Therefore all tests for increased muscle tone have to be subjective and based upon the findings of the examiner. However, increase in knee jerks and functional ability, as well as the actual feel of the muscle, is considered a reliable clinical test.

The site of action of strychnine, according to the books on pharmacology, is in the spinal cord. Unquestionably, in the large dosages that we are giving one might expect also a central cortical effect. It is interesting to note that in the cases of muscular dystrophy where the lesions are presumed to be in the muscles per se, toxic doses of strychnine (up to 1/30 grain [2 mg.] three times a day) did not cause any increase in muscle tone, tremors, startle reflex, or convulsions.

One wonders whether it is possible to precipitate a convulsion in a far advanced case of muscular dystrophy. In our experience, no such case of the occurrence of muscular dystrophy with convulsions comes to mind. Our experience with the final case of cerebral palsy, which has been repeated in several other such cases, indicates that this drug is effective in increasing muscle tone in patients with cerebral palsy in whom hypotonia is an outstanding symptom. If this is the case, the drug may be valuable as an adjuvant in the treatment in these cases, particularly so far as standing, walking, and other supportive activities are concerned. Whether such trained improvement will be sustained after the drug is stopped is still a matter of conjecture.

A Rare Type of Tracheoesophageal Fistula. Dr. Maxwell P. Borovsky.

Diaphragmatic Hernia. Dr. John L. Keeley.

Diagnosis of the Taussig-Bing Heart. Dr. Benjamin M. Gasul, Dr. Don L. Fisher, and Dr. Howard Weiss.

The patient, a 28-month-old boy, has been cyanotic since birth. He is too weak to walk. He had pertussis at the age of 8 months and measles at 18 months. The mother was not ill during pregnancy.

Physical examination revealed a somewhat undernourished, cyanotic baby boy weighing 19 lb. (8.6 kg.). There was moderate clubbing of fingers and toes. Femoral pulsations were normal. The cardiac apex was in the fifth interspace, about 1 cm. to the left of the midclavicular line. A grade 3 systolic murmur was heard all over the precordium, and the second pulmonic sound was louder than the second aortic.

Fluoroscopic and roentgenographic examinations in the posteroanterior, the right anterior oblique, and the left anterior oblique views revealed a moderate enlargement of the transverse diameter of the heart, a convexity in the pulmonary area, exaggerated vascular pulmonary markings, and an enlargement of the right auricle and of the right ventricle. A barium esophagram showed a left aortic arch and anterior displacement of the esophagus in the region of the aortic arch in the left anterior oblique view, probably a double aortic arch.

The electrocardiogram (15 leads taken) showed right auricular and right ventricular hypertrophy. The angiocardiogram showed simultaneous visualization of the aorta and of a large pulmonary artery.

The hemoglobin was 100% (15 gm.); the red blood cell count 6,930,000; the color index 0.72, and the white blood cell count 7,500, with a normal differential blood count.

Catheterization of the heart revealed the following data:

	Blood Oxygen Contents (Vol. %)	Pressures (S/D, Mean)	
		Mm. Hg	Normal
Superior vena cava	8.09	4	5-10
Right auricle	8.04	4	5-10
Right ventricle	8.10	73/2 (43)	25/5 (12)
Right ventricle	10.67	.....	.....
Right carotid artery	8.83 (43%)	70/48 (63)	.....
Left femoral artery (crying)	3.99 (18%)	.....	.....
Left pulmonary artery	15.52	79/51 (64)	25/10 (18)
Arterial oxygen capacity	20.40	.....	.....
Hemoglobin	15.0 gm.	.....	.....

Calculated Blood Flows:

Pulmonary flow: 1.30 meters per minute; index 3.72 liters per minute per square meter.

Systemic flow: Could not be calculated in the presence of such a small arteriovenous difference. The figures would be meaningless.

Spot films of the catheter illustrated the aorta coming off the right ventricle, and the catheter entered the pulmonary artery either directly from the right ventricle or through the interventricular septal defect. The oxygen saturation of the pulmonary artery is much higher than that of the aorta.

Dr. Taussig and Dr. Bing were the first to report a case of complete transposition of the aorta with the pulmonary artery overriding both the right and the left ventricle (*Am. Heart J.*, **37**:551, 1949). We believe that our findings in this case demonstrate the presence of a Taussig-Bing heart.

Pitfalls of Acetylsalicylic Acid Medication. DR. WILLIAM S. HOFFMAN.

At Cook County Hospital we have three types of patients with acetylsalicylic acid poisoning, five or six or more a year. These are, first, patients who attempt suicide by taking a single large dose of acetylsalicylic acid, usually women. The second group are small children who take a large number of tablets thinking it is candy. The third group includes chiefly infants and children who receive an overdosage of acetylsalicylic acid medication. The dosage in adults is figured more accurately than for children.

Acetylsalicylic acid was invented to avoid the gastric irritation of salicylate therapy. It is not completely hydrolyzed in the intestines, but after one half hour or more practically all that is in the blood is in the form of salicylate. The excretion of salicylate under normal acidity of urine is difficult and the kidney reabsorbs some of it. The bulk of salicylate found is in the form of conjugates of salicylate formed by the kidney and liver and excreted through the tubules. This so-called detoxification represents conversion of substances difficult to excrete into more easily excretable substances.

In the infant, excretion is less because of the well-known immature function of the kidneys. It is probably poor also because conjugation is an attained function in the body and is not accomplished so well by the infant. Thus there is more difficulty in getting rid of salicylates in the infant than in the adult. It takes about 24 or 30 hours to excrete a single dose of aspirin. The plasma level at the end of four hours is about 4 mg. per 100 cc. when one grain (60 mg.) is given to an infant. With the modern tendency to give drugs every four hours, a second dose of one grain to an infant will cause a pyramiding of the blood level, so that at the end of 24 hours the blood level may be 20 or 24 mg. per 100 cc. If the doses are maintained at the same rate, obviously extreme toxicity will develop in two or three days. Most pediatricians are not acquainted with this phenomenon and do not realize that this pyramiding takes place.

One baby seen in the clinic was sent home with a prescription of one-grain aspirin tablets. The mother gave three 5-grain (300 mg.) tablets. The blood level was found to be 45 mg. per 100 cc. Our experience is that every one with a level of 35 mg. per 100 cc. has some degree of salicylism. With 45 mg. per 100 cc. there are tinnitus, malaise, headache, and alkalosis. In the infant we see marked hyperventilation and respiratory alkalosis. The urine tends to become alkaline.

Treatment is for the most part expectant. Fluids are encouraged to promote diuresis. It is only a matter of hours before the child is out of danger in most cases, but there may be shock and prolonged intoxication. We originally recommended giving bicarbonate and lactate to help excretion of salicylate, but after one death we have become more timid and give glucose solution, which is usually satisfactory. Theoretically, treatment should be directed toward reversing the respiratory alkalosis by giving carbon dioxide with oxygen. We have done that but would not recommend it until the treatment has become standardized. In mild cases one may give alkali, but it is not necessary, and in severe cases to increase the alkalosis might make the difference between life and death.

Infection and the Allergic Child. DR. ETHEL M. DAVIS.

At the Cook County Children's Hospital certain children have been repeatedly admitted with severe asthma, with and without upper respiratory infections. In the absence of fever and other evidences of infection, the child is given epinephrine in the examining room and not admitted.

In order to study these children and others with repeated admissions for severe allergic manifestations, the Children's Allergy Clinic was set up in January, 1945. This clinic also serves

to train some of the pediatric residents and to familiarize them with the procedures of allergic workup and management.

What is the relation of the upper respiratory infection to the allergic manifestations in these children? There are two schools of thought. We feel that the upper respiratory infection acts as a precipitating and/or aggravating factor which throws the child out of allergic balance; i. e., allergic symptoms develop. The other school promulgates the idea that the allergic manifestations such as asthma is a reaction of hypersensitivity to the infecting micro-organisms. Because the child is sensitive to the organisms in his nose, tonsils, accessory nasal sinuses, nasopharynx and/or bronchial tree, asthma develops. Such children are subjected to removal of tonsils and adenoids, irrigation of sinuses, and irradiation of the nasal mucosa. Treatment with respiratory vaccines, stock or autogenous, is undertaken. The asthma continues or is aggravated, especially following nasopharyngeal surgery such as removal of tonsils and adenoids. We feel that in no instance is the infection the specific cause. A reaction to micro-organisms such as occurs to ragweed pollen is unknown.

Children referred from the hospital wards are completely examined and tested. Any person with asthma is entitled to a complete workup, including x-ray of the chest. The more substances to which he is sensitive that are identified, the more effective is management directed to control of the asthma.

Both scratch and intradermal tests are done. Management is as follows: Substances to which the child is sensitive are removed from the environment if possible. To those which cannot be removed, such as pollens, molds, and house dust, he is rendered less sensitive by hyposensitization. As a result of treatment with extracts of the substances to which he is sensitive, he becomes less sensitive, so that he can come in contact with these substances without having symptoms. The results of this type of management have been brilliant. Not only is the asthma brought under complete control, but the child has fewer colds and upper respiratory infections. Children gain rapidly in growth and development once the allergy has been controlled.

Treatment should be undertaken if possible before complications and irreversible changes occur, such as infection of the nasal sinuses with or without polyp formation and chronic bronchitis and emphysema. After the allergic manifestations have been brought under control, many children remain free from symptoms over long periods of time without further treatment.

Craig D. Butler, M.D., *President*

Regular Meeting, Feb. 19, 1952

The Allergic Cripple. Dr. Townsend B. Friedman (by invitation), Dr. William C. Grater (by invitation), and Dr. Robert A. Stier (by invitation).

Psychological Services for Children in Chicago. Dr. Gustave F. Weinfeld, Highland Park, and Dr. Freda Kehm (by invitation).

Last year questionnaires were submitted to 65 of the larger hospitals and clinics in the city of Chicago. Inquiries were also made of other important public agencies concerning their facilities for providing for the psychological care and treatment of children.

This report is an attempt to set forth the recognized goals of this type of care as expressed in the policies of such groups and agencies as the 1950 White House Conference and the Welfare Council of Metropolitan Chicago.

The data procured from the 65 reporting hospitals and other clinics and agencies have been tabulated, correlated, and interpreted.

A wide variety in quality and quantity of services is offered by the reporting groups. As in all studies of this sort, there can be no claim for the statistical accuracy of the various data because of the variety of standards employed by those who furnished analyses of their services. For example, a part-time psychiatrist or psychologist may be one who gives only 2 hours a week in one clinic or 20 hours a week in another. Case loads were equally difficult to determine.

This study may serve as a preliminary survey of current psychological services in a large limited urban area. It may also provide future clues for enlarging, improving, and correlating this important phase of child care.

Congenital Heart Disease Following Maternal Rubella. Dr. Stanley Gibson and Dr. Katherine C. Lewis (by invitation).

This paper was published in full in the March, 1952, issue of the American Journal of Diseases of Children, page 317.

DISCUSSION

Dr. Archibald Hoyne: There is a question that arises in connection with this problem of rubella, not merely because of the possibility of some deformity, but as a consequence of the diagnosis. A few years ago, Fox, in Milwaukee, made a survey, as a result of which he came to the conclusion that there were no more deformities in the group in which there was a history of rubella than in the normal run. At the present time, when the average physician detects a blemish in a pregnant woman he thinks that she may have German measles, which throws quite a responsibility on the physician called upon to make the decision. One Chicago obstetrician did not specify the number, but said that he had seen a great many cases of German measles during pregnancy, and none of the infants had any defect. The question is, does German measles always cause a deformity, or is the deformity due to German measles?

In the last year or so I have seen six or seven instances where women had German measles, that is, it was thought that they had it. In several cases it seemed certain to me that they did not have it, and in others it seemed that the diagnosis was correct. On two occasions the obstetrician was indignant because I would not make a diagnosis of German measles. I do not know whether anyone here has seen a child with a deformity and known that the mother had German measles. I wonder if there is any question with regard to the accuracy of the history on these patients. If a child has a deformity, the mother wants some explanation or excuse. The history is usually obtained four or five years after the birth of the child, and I wonder whether the diagnosis is correct, or whether the mother has been reading the magazines and has been influenced in such a manner. It is difficult to believe that the investigation made by Fox could come out as evenly as it did, but I am sure that according to that report German measles was responsible for no more cases of deformity than are ordinarily observed.

Dr. Benjamin M. Gasul: We did not make a study of these cases in our series, but I can recall four or five children with congenital malformation of the heart that was probably due to the mother's having rubella during the first trimester of her pregnancy. All these patients had a noncyanotic type of congenital malformation of the heart.

Dr. Katherine C. Lewis: In our study no correlation was found between congenital heart disease and diabetes. The answer to the question as to how many mothers in such a group can be expected to have had rubella could be obtained only by a separate study.

In an exhaustive review of the literature, the article by Fox and Bortin, in 1946, presented the only contrary evidence on this question of rubella. They discussed the question of therapeutic abortion in women who had rubella in the first two months of pregnancy and came to a negative conclusion. Dr. Hoyne mentioned the difficulty of the diagnosis of rubella and mentioned that perhaps the diagnosis was not properly confirmed. He also suggested that the use of therapeutic abortion might be abused. Only those cases in which the diagnosis had been verified were considered in our study. One may also mention that in Australia, where the syndrome was first noted, an epidemic of rubella gave the clue to the cause of the anomalies. The National Society for the Prevention of Blindness, with the American Academy of Pediatrics, made a study of 132 mothers who had had rubella in the first trimester of pregnancy. Only 18 of the offspring were normal; 67 of the 132 had congenital heart disease. In 1946, Swann, of Australia, reported that in his study 100% of the women who had had rubella in the first two months of pregnancy and 50% of those who had had rubella in the first three months gave birth to children with anomalies.

A child who is a deaf-mute, with congenital cataracts, congenital heart disease, and possibly also mental retardation, is a seriously handicapped individual. To prevent such instances, the disease in the mother must be prevented. Therefore one must view seriously the occurrence of rubella in the pregnant woman.

Craig D. Butler, M.D., *President*
Regular Meeting, March 18, 1952

Bronchial Asthma in Children: Treatment and Results—A Thirty-Year Study. DR. A. ALVIN WOLF (inaugural thesis).

Uterocele. DR. KNOWLTON E. BARBER (by invitation).

Transabdominal Pneumoperitoneum as an Aid in the Diagnosis of Sex-Endocrine Disturbances. DR. RALPH H. KUNSTADTER, DR. HENRY S. GUTERMAN (by invitation), and DR. ALEX S. TULSKY (by invitation).

It is the purpose of this paper to show how pneumoperitoneum can be adapted to children and how, in various sex-endocrine disturbances, it has aided in establishing or verifying a clinical diagnosis. To the best of our knowledge, this procedure has not been described in pediatric literature, and in gynecologic writings no mention is made of its use in children.

A transabdominal pneumoperitoneum was performed in 16 cases, 6 of which are presented to illustrate the value of the technic. It would appear that the procedure may be of particular value in establishing the presence of pelvic anomalies and other pathologic conditions, more especially when a clinical diagnosis cannot be established without laparotomy or when laboratory data are inconclusive.

Prior to pneumoperitoneum all patients receive a diagnostic workup, the various laboratory procedures being performed according to individual problems presented. The technic of the procedure consists of introducing a measured amount of CO_2 into the peritoneal cavity and then visualizing radiographically the outlines of the pelvic viscera. A special radiographic table designed by Dr. Irving F. Stein and Dr. Robert Arens is used.

The 16 cases thus studied included ovarian agenesis, precocious puberty, hypopituitarism, hypovarianism, cystic ovary, and intersexuality. Early diagnosis in such cases is desirable in order to plan future medical and/or surgical management, with the purpose of prevention of profound emotional and physical damage.

DISCUSSION

DR. IRVING F. STEIN: I consider it a privilege to hear Dr. Kunstadter and I congratulate him upon his fine and valuable presentation.

My own experience has been chiefly with adults and in the past 30 years I have used this method in about 3,000 cases. I have had a little experience in infants and have studied a number of juveniles.

Dr. Kunstadter mentioned the fact, and I wish to emphasize it, that pneumoroentgenography may be the means of avoiding an unnecessary operation, not only in children, but also in adults. It is a simple and safe method of diagnosis.

As a point of technic, the bladder must be emptied before pneumoperitoneum, usually by means of a catheter.

I believe that x-ray in gynecology and in pediatrics is just as valuable as is x-ray in chest and gastrointestinal work, and until recently it has been bypassed by many doctors. I have had a great deal of gratification from my work, for I feel that this method has given me most valuable information.

DR. MATTHEW M. STEINER: We have used this procedure recently and have found it valuable in some cases. I certainly agree with Dr. Kunstadter and Dr. Stein that it will avoid operative intervention, particularly in ovarian agenesis. Recently we had two cases in which it was difficult to differentiate from hypopituitary dwarfism. Without delay for laboratory studies, the pneumoperitoneum did indicate that one patient had ovaries and the other did not. In pseudohermaphroditism one must visualize the ovaries to see if there is a mixed type. While pneumoperitoneum may be a valuable adjunct, it is often necessary to do a biopsy on the ovaries.

I feel, as Dr. Kunstadter does, that we should adopt this procedure more widely. Certainly pediatricians in good hospitals can do it; it is a simple procedure.

George F. Munns, M.D., *President*
Regular Meeting, Nov. 18, 1952

The Management of Diabetic Acidosis in Children. Dr. George M. Guest (by invitation) (from Children's Hospital Research Foundation and the Department of Pediatrics, University of Cincinnati College of Medicine), Cincinnati.

The metabolic derangements which characterize the development of ketonemic acidosis and coma are discussed with a view to a rational plan of therapy. Theoretically, the diabetic patient should never go into ketonemia, acidosis, and coma if he is under proper management. Ketonemia and acidosis are primarily due to insulin insufficiency rather than to overingestion of carbohydrates. With lack of insulin there is impaired glycogenesis and loss of glycogen from the liver. The liver cells burn more fat, leading to overproduction of ketone bodies and production of ketone acids, resulting in decreased alkaline reserve, decreased bicarbonate in the blood plasma, and lowered pH. The state of acidosis per se leads to increased catabolism, liberation from the cells of labile intracellular constituents into the plasma, which are lost into the urine. The increased urinary excretion of phosphates, potassium, and sodium leads to increasing acidosis, inability of the body to retain water, and aggravation of the progressively developing state of dehydration. Dehydration itself is an important factor in the development of acidosis.

The loss of electrolytes aggravated by the state of acidosis is a principal factor in the development of dehydration, the additional factor being diuresis. Hemoconcentration, tissue damage, and cellular damage become important factors in relation to prognosis. The factor most closely correlated with mortality has been shown to be not hyperglycemia or severity of ketosis, but the degree of acidosis as measured by the low pH and the length of time it has been low. With long-continued acidosis there is an element of irreversible tissue damage. What that damage is we do not know, but the development of unconsciousness is certainly correlated with diminished cerebral oxygen uptake.

Recovery is dependent upon rehydration, effective insulin action and the attendant anabolic effects of insulin, administration of parenteral fluids (salt solution, alkalies, dextrose), supportive treatment by chemotherapy in the presence of infection, vitamins during the recovery period, and orally administered fluids. Everyone is agreed that administration of insulin is the important consideration. There is debate as to the dosage; some favor huge initial doses, and others give relatively small doses at short intervals. Restoration of efficient circulation by increasing circulating blood volume is also of primary importance. The administration of alkalies has also been controversial, owing, probably, to the abuse of its use in preinsulin days. Even with severe degrees of acidosis it is necessary, in most instances, to raise the CO_2 by only 15 vol. % to bring the pH of the arterial blood to normal, thus restoring a favorable environment for enzymatic processes of cellular metabolism.

The question of dextrose solution administration has also been controversial; most people feel it should not be given until the blood sugar has fallen to normal. It is important not to give dextrose, particularly hypertonic dextrose solution, until insulin action becomes effective. When this occurs the administration of dextrose may be beneficial even before the blood sugar has fallen to normal.

The sequence in therapy is as follows: First, insulin in appropriate doses, and we prefer repeated small doses rather than initial huge doses of several hundred units. There is evidence that insulin may be destroyed in the acidotic patient, or excreted; thus, repeated small doses give the promise of more effective continuous action. Next is the administration of salt solution in appropriate amounts to improve the circulation. Then the administration of alkalies to bring the pH of the blood to normal, and, after the third or fourth hour, the administration of dextrose in 5% or even 10% solution, to accelerate glycogenesis. We now know the important role played by potassium in the postacidotic period. The question is how much to give. The amount must depend upon the ability of the tissues to absorb it, and the state of the patient with regard to previous depletion. Much research needs to be done on the indices that govern a rational plan of potassium dosage, and until this has been determined it would be best to give minimal amounts, with careful observation of the electrocardiographic findings.

DISCUSSION

DR. M. DAVID ALLWEISS: As Dr. Guest stated, diabetic coma is preventable for the most part. The education of the diabetic patient as to his insulin requirements is the responsibility of the physician primarily.

Insulin given subcutaneously to patients in shock is sometimes not effective, and often it is best given intravenously. One should determine the serum protein as quickly as possible, keeping in mind that a normal serum protein in the presence of hemoconcentration seen in severe dehydration merits the use of serum plasma and whole blood transfusions.

Dr. Guest mentioned the work of Mirsky in which he produced acidosis in dogs, then gave them dextrose, and showed that the values for blood acetone fell as those for blood sugar rose. The kidneys were tied off in those dogs, and that probably explains why the experiment could not be translated to humans. Some work was reported two years ago from England in which dextrose was administered to patients in diabetic coma, and the mortality rate was high in contrast to that in patients given saline only. All of those patients had received sufficient insulin. The patients who received dextrose lost 43.3 % of the fluids administered, in contrast to those receiving saline, who lost 18% of the fluids in the urine. Thus diuresis caused by dextrose can be dangerous.

DR. JAMES B. HURD: Dr. Guest's approach to the problem of electrolyte imbalance in diabetic coma is interesting. We have measured this primarily from the physiologic viewpoint —the computation of electrolytes in the serum and the output of electrolytes in the urine. The study of the electrolyte components and phosphates, organic and inorganic, in the blood cells is a step in the direction of ascertaining what is going on in the cells. During diabetic coma we cannot measure in either animals or humans the exact concentration of and the changes in electrolyte balance within the cells.

I should like to take issue with Dr. Guest on his routine of frequent small doses of insulin. We have followed the method of Dr. Woodyatt and Dr. Colwell of giving large doses initially, following the known physiologic pattern of insulin action within the body over a period of four to six hours. It seems to us that giving smaller amounts every hour or half hour merely confuses the picture. If in the first hour we underestimate the dose, then repeat the dose in another two hours and, by chance, still underestimate it, we have lost two important hours in instituting adequate insulin therapy. We watch the patient carefully. Within three to four hours we should begin to see clinical improvement, and, if we do not, we repeat the insulin dose, possibly increasing it.

I think Dr. Guest's outline of fluid therapy is excellent. The administration of dextrose early serves not only to increase diuresis and increase electrolyte output, but also increases the confusion. Other than the urine test we have no laboratory means, at least in the average hospital, of determining the result of treatment. If we give dextrose in the first few hours there is a heavy glycosuria which persists for four or five hours.

I should like to ask Dr. Guest his criteria for the effectiveness of therapy.

DR. ARTHUR R. COLWELL, Evanston: Dr. Guest has given us an orderly reasonable physiologic and chemical explanation of diabetic acidosis and coma as seen clinically and experimentally. This leads to an explanation of the reversal of that picture, the correction of it by treatment. It seems as though the controversy to which he referred, between the clinician and the investigator, is becoming resolved. It is interesting that it took the flame photometer to end the controversy concerning the early use of dextrose. Some of us learned long ago, by counting the drops of urine as they fell from the catheter and comparing this number with the number of drops of intravenously administered dextrose as they dripped into the patient, that water left the body more rapidly than it was put in. When we did not use dextrose we could easily cause retention of fluid. Watching the urinary output is a very simple way of determining when dextrose should be given in the treatment of diabetic acidosis. Blood sugar values or quantitative data of any kind are not needed. When the extreme diuresis begins to lessen, as observed carefully by catheter, then is the time to give dextrose.

We feel that small doses of insulin given over a period of time deprive the patient of insulin which he needs immediately, and we favor administration of an initial large dose.

DR. ALVAH L. NEWCOMB, Wilmette: This story is almost a tragedy of errors. An 8-year-old girl came into the Children's Memorial Hospital dispensary with a recognized

acidosis and was thought to have acute tonsillitis, probably scarlet fever. The intern obtained a typical history of diabetes mellitus, but the urine was sent to the laboratory, and a 1+ value for sugar was reported. The patient was treated vigorously with fluids, alkalies, sulfadiazine, and penicillin. At about 7 p. m. Kussmaul respirations developed, gross blood was discovered in the urine, and oliguria was noted. The resident recognized the diabetes and immediately started therapy. The child was desperately ill, and we were not sure that she would recover. The following morning ketonuria and glycosuria had subsided. Hematuria persisted for several days. All sulfonamides are contraindicated until the dehydration of diabetic coma is relieved.

About two years ago we treated a 13-year-old girl with diabetic ketosis. The chemistry returned to the normal level but at the time she did not wake up. Finally she awakened, and it was learned that she had taken a lot of tripelennamine (elixir pyribenzamine®) citrate for a cold the day prior to the onset of diabetic ketosis.

DR. W. S. HOFFMAN: I should like to point out that the fall in serum potassium concentration which occurs during treatment of diabetic acidosis is only partly due to the uptake of dextrose by the cells. It is also due to dilution of the extracellular fluid, to increased renal excretion, and to improvement of the cellular oxidative processes. In spite of the fact that the patient will have lost large quantities of potassium by the time he is seen in coma, the serum potassium level at this stage is often much higher than normal. The net cellular transfer of potassium at that moment is away from the cells rather than into them. If potassium is offered the body at this stage, the serum potassium will rise to dangerously high levels. However, if enough saline is given to improve the patient's general condition and renal function, with an adequate urine output potassium chloride can be safely added to the intravenously administered fluids in amounts of 2 to 3 gm. per liter. The amount that can be given in the first 24 hours is never great enough to repair the deficit completely, or to bring the serum potassium level to normal. Often at the end of 24 hours, the serum potassium level is still around 3 mEq. per liter. But if no potassium is added to the parenterally administered fluids the level might be below 2 mEq. per liter, and the patient would be in a serious condition

DR. GEORGE M. GUEST, Cincinnati: Dr. Hoffman mentioned an important point with regard to changing levels of potassium and of inorganic phosphorus in the blood at different stages during recovery from diabetic acidosis. The direction of movement of these elements between cells and extracellular fluids at different times is also an important factor. The administration of potassium in the presence of hyperpotassemia may have deleterious, even dangerous, consequences. While potassium is escaping from the cells into the plasma anything that promotes diuresis will aggravate the losses into the urine; thus early administration of glucose solution is contraindicated. On the other hand, the direction of movement of intracellular elements changes after anabolic processes are restored, when the cellular uptake of potassium and phosphorus becomes a dominant feature in the metabolic balance. At that point the urinary excretion of potassium, nitrogen, phosphorus, and calcium diminishes abruptly. In the early hours of treatment with insulin and salt solution alone, while acidosis persists, potassium continues to escape from cells. I have insisted that quick correction of the pH of the plasma by administration of an alkali is important and may be a critical procedure in the treatment of the severest cases of coma.

Dr. Newcomb rightly suggests that the sins of omission, in failure of diagnosis, may be as important as sins of commission in the treatment of diabetic acidosis in young patients. In our hospital also, diabetic acidosis has gone undiagnosed too long when the trouble was thought to be merely "respiratory disease."

Dr. Colwell touched on a much debated question, weighing the advantages of initial large doses of insulin versus repeated small doses. The scheme for initial dosage in our hospital is roughly as follows: For ages up to 2 years, 10 to 20 units; 5 years, 15 to 30 units; 10 years, 30 to 75 units; 15 years, 40 to 100 units. In the presence of severe infection and severe acidosis those doses may be doubled; such doses are repeated at one- to four-hour intervals until ketosis is eliminated. Note that the effect of insulin given intravenously is rapid, with principal effects normally apparent in the first 30 to 40 minutes. Some investigators claim that insulin may be destroyed in the body more rapidly than normal, under conditions of severe infections. Repeated small doses may insure a better continuity of the maximal effects of insulin.

Replying to Dr. Hurd, the principal criteria for insulin effects are as follows: (1) decreasing blood sugar, demonstrable if glucose is not administered early; (2) fall of inorganic phosphorus and of potassium in the plasma, and (3) elimination of ketosis. I should like to mention that we have used the acetest® (tablets containing aminoacetic acid, disodium phosphate, and sodium nitroprusside) of the Ames Company, Inc., and other "acetone test powder" in following the decrease of plasma ketones. Blood drawn by skin puncture into capillary tubes (volume 0.1 or 0.2 cc.) sealed and centrifuged, will yield a sufficient quantity of plasma for the test. Drops of plasma are diluted with drops of water, 1:2, 1:4, 1:8, etc. Single drops of the diluted plasma added to the acetest® tablets produce shades of purple varying according to the concentration of ketones. Drops of standard solutions of pure acetone similarly added to a parallel series of tablets afford a quantitative basis for comparison and estimation of concentrations of ketones in the plasma. Acetoacetate in the plasma yields a color roughly five times the intensity of that given by pure acetone. Ketones thus demonstrable disappear from the blood long before they disappear from the urine.

Dr. Allweiss is, of course, right in mentioning the importance of serum protein levels as an index for the inclusion of plasma or whole blood transfusions in the treatment of diabetic coma; but in children this is less often indicated than in adults. I believe we have not used blood transfusion in the treatment of diabetic coma in any child during the past 10 years. Transfusions are, however, employed often on the adult medical service of the general hospital, where older patients are often admitted after 48 hours of coma, suffering the effects of shock and hemoconcentration as major features of the crisis. Mild degrees of acidosis and coma of short duration should respond quickly to judicious treatment with insulin and the usual parenteral fluids without the use of blood or plasma.

Obituaries

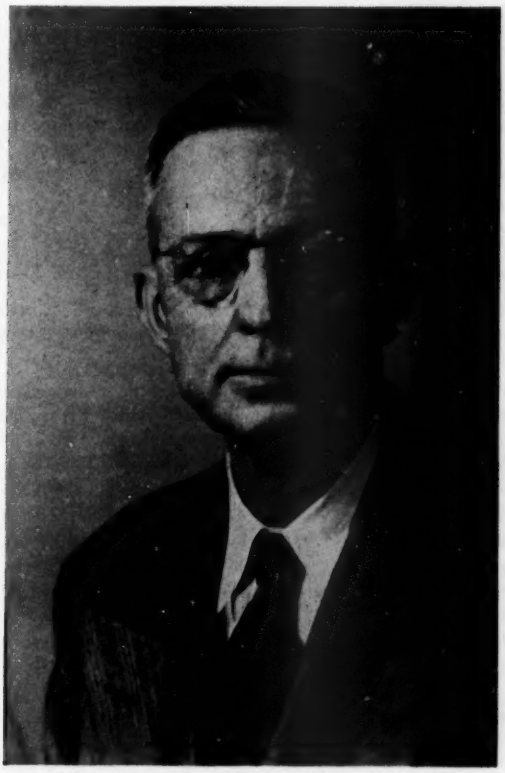
PHILIP CHARLES JEANS, M.D.
1883-1952

WITH THE death of Philip Jeans on Oct. 22, 1952, medical education in general and pediatrics in particular has lost one of its outstanding contributors. Philip Charles Jeans was born in Hillsboro, Ohio, on Jan. 3, 1883. He received his A.B. degree from the University of Kansas in 1904 and M.D. from Johns Hopkins University School of Medicine in 1909. Called to Washington University by Dr. McKim Marriott in 1913, Dr. Jeans was successively assistant instructor, associate instructor, and associate professor of pediatrics on that faculty. In 1924 he was appointed professor of pediatrics of the University of Iowa Medical Department, which post he held until his recent death. At the time of his death he was serving as a special consultant and lecturer for the World Health Organization program for appraisal of nutrition in the Central American republics.

He is survived by his widow, Grace (Whittier) Cushing Jeans, whom he married in 1914. Trained in the Howland tradition and with the discipline passed on through Marriott, Philip Jeans was well prepared for his pediatric professorship with his studies on nutrition. He had the rare gift of translating sound investigation to realistic clinical adaptation. As such, he became a scientific authority on nutrition, but equally vigorous in his public services for the Council on Foods and Nutrition. Early appreciating the proper use and place of vitamins, particularly vitamin D, and the simplified methods of infant feeding, he was the logical author of the revision of Marriott's textbook.

Besides his membership on the American Medical Association Council on Foods and Nutrition, Dr. Jeans was a member of the Food and Nutrition Board of the National Research Council. In 1940 the American Institute of Nutrition presented him with the Borden award. He was a member of the American Pediatric Society, the Northwestern Pediatric Society, Central States Pediatric Society, and the Society for Experimental Biology and Medicine.

Behind the quiet reserve so characteristic of the man was the authoritative wisdom that made him the fine teacher and reliable investigator that he was. The unique Department of Pediatrics at the University of Iowa was to a great extent his development, and a notable group of students, investigators, and disciples can be traced to his stimulation and leadership.



PHILIP CHARLES JEANS, M.D.

1883-1952

Abstracts from Current Literature

Biochemistry; Bacteriology and Pathology

EXPERIMENTATIONS ON "SELF-INTERFERENCE" OF THE MM-VIRUS. O. VIVELL, Ztschr. Kinderh. **70**:113 (No. 2) 1951.

The author wanted to investigate the "self-interference" of the encephalomyocarditis group of viruses which are pathological for man and ape and fatal to mice, etc. If a certain small amount of virus is injected under the skin of the mouse and followed a few hours later by a larger amount, which if given as first injection would be fatal, no fatal reaction is produced. The author points out that the portal of entry must be the same and the dosage and timing correct to obtain this counteraction, and he feels that this phenomenon may offer the explanation for the fact that in epidemics few individuals remain free of the disease.

GLASER, Jerusalem.

Metabolism; Infant Feeding; Milk and Other Foods

DETECTION AND TREATMENT OF NUTRITIVE FAILURE IN CHILDREN. TOM D. SPIES; SAMUEL DREIZEN; GEORGE S. PARKER, and DONALD J. SILBERMAN, J. A. M. A. **148**:1376 (April 19) 1952.

Growth and nutrition with all their complexities are little understood. A case history is reported of a woman receiving a diet inadequate in folic acid during her six pregnancies. During each pregnancy severe anemia developed, which, continuing afterward, even caused severe anemia and growth failure in her breast-fed infants. Adequate folic acid in the mother's diet cured the mother's anemia and restored adequate milk production. In addition, the anemia in the infants was corrected without the infants' being given any supplementary vitamins directly.

Cortisone and corticotropin improved the appetite and weight gain in a fatal case of leukemia.

A large, stocky but "fractious" boy whose diet consisted of cornbread, biscuits, fat pork, sugar, and occasionally turnip greens, corn, tomatoes, and berries, was found by biologic assay to have a diet lacking in casein, minerals, and vitamins. For three years, from the time that the patient was 8 years 9 months of age, a dietary supplement of dried skim milk (equivalent to 3 qt. [3 liters] per week) was added, which resulted in steadier growth and better concentration and behavior.

The authors believe that these changes are not irreversible.

KIMBALL, Evanston, Ill.

THE TREATMENT OF SEVERE NUTRITIONAL DISTURBANCES IN INFANTS WITH ANTIBIOTICS. E. MARTISCHNIG, Ann. pædiat. **179**:168 (Sept.) 1952.

A survey is given of the various opinions regarding the importance of the intestinal flora in diarrhoeic diseases of infants; its domineering influence in the pathogenesis is made clear. The importance of the "pathogenic" coli strains, particularly the saccharose-fermenting ones and the strains 111 B_4, and 55 B_8 is shown. The up-to-date results of antibiotic treatment with streptomycin, aureomycin, hydrochloride and chloramphenicol (chloromycetin®) are reviewed. The necessity of exact sensitivity tests is emphasized, which alone would vouch for optimal therapeutic results. The author's experience consisted of the treatment of 125 infants, 67 with intoxication and 58 with severe enteritis. One hundred and twelve children were cured, and 13 died. The 13 cases in which death occurred are extensively reported, and the deduction is made that the prognosis is especially unfavorable in children under 3 months, in those who received no or insufficient quantities of breast milk and, finally, where a complicating illness coexisted.

FROM THE AUTHOR'S SUMMARY.

COMPARATIVE INVESTIGATIONS OF THE WATER-COMBINING POSSIBILITIES OF VARIOUS STARCHY FOODS. OLEG SELAWRY, Helvet. paediat. acta **5**:246 (June) 1950.

A number of proprietary starch preparations were examined for gel formation, water-binding capacity and viscosity. Their respective value in infant feeding is discussed in the light of these physical characteristics. The following preparations were examined: (1) potato starch ("soldor," Nestlé); (2) desiccated ground carrots ("elonac," Guigoz); (3) corn flour ("maizena"); (4) desiccated, ground, and roasted fruit pulp of the carob plant with an admixture of "soldor" (15%) and coca (5%) ("arobon," Nestlé), and (5) the flour of dried carob seeds ("nestargel," Nestlé).

A gel prepared from a 10% solution of "arobon" or "elonac" binds water poorly, the solution being of low viscosity; gel formation is practically absent. Therapeutically, these properties are reflected in a reduction of the volume of the stools and their tendency to become firm and dry. "Elonac" has a better water-binding capacity than "arobon"; hence it produces bulkier stools. The high NaCl concentration of the latter preparation, however, is likely to produce edema in the baby once the water deficiency has been made good.

Corn flour at 1% concentration behaves in a similar manner, though at higher concentrations it has a much greater capacity for gel formation, and potato starch is even better as far as the latter property is concerned. A 2 (to 3)% concentration is optimal in thickening bottle feeds.

"Nestargel" has by far the greatest water-binding capacity and viscosity; its gelling capacity is not as good as that of corn flour. These physical properties make the carob-seed powder the ideal thickening for bottle feeds, since a high degree of viscosity can be obtained at low concentration; 0.5% may be effective and 2% should not be exceeded when conditions such as aerophagy, food regurgitation, and some forms of vomiting are being treated by dietary means.

FROM THE AUTHOR'S SUMMARY.

POOR NUTRITION AMONG INFANTS IN THE DISPENSARY: THE CAUSES AND AGE OF ONSET. JULIAN SANTAMARIA PULIDA, Acta pediat. españ. **98**:162-172 (Feb.) 1951.

Dr. Pulida studied some 75 patients seen in the free dispensary and classified them according to nature of first illness, social condition, and causes for their poor nutrition. All were tested for syphilis and tuberculosis. They were also classified as to breast or artificial feeding.

Rarely was there any specific disease such as syphilis, tuberculosis, or malaria, although there were a few cases of each.

His graphs show that the largest number of malnourished infants began their troubles in the first month. The peak was reached between the second and third month of life.

There did not seem to be any difference between breast-fed and artificially fed babies. Most of the children came from very poor homes, although there were some in the high economic strata whose mothers appeared to be indifferent or just said that the baby was naturally delicate.

The author believes that any health center, however modest its set-up, should have a visiting nurse who can educate the mother in the essentials of infant feeding.

In 52% of the cases poor nutrition was due to poor and inadequate feeding, including a deficiency of vitamins from the common sources; acute infections, including diarrhea (which may also be due to poor feeding), accounted for 43%. In 4% of the cases poor nutrition was due to constitutional causes, including syphilis, tuberculosis, and malaria.

ALLEN, Santa Fe, N. M.

INFLUENCE OF ULTRAVIOLET RAYS UPON THE PROTEIN OF MILK SERUM. T. F. HELLBRÜGGE, L. SCHEID-SEYDEL, and H. MARTIN, Ztschr. Kinderh. **70**:1 (No. 1) 1951.

Milk serum was obtained by centrifugation, whereby the fat remained on the surface, the casein settled out, and the serum in the middle layer was used for the experimentation. Electrophoretic analysis and the coagulation reaction of Weltmann were used to analyze the changes which had occurred after ultraviolet irradiation. The earliest changes were discovered after 20 seconds of irradiation. Since for prophylaxis of rickets irradiation for 0.5 second is usually applied, no measurable influence upon the protein should be exerted by this prophylactic treatment of the milk.

GLASER, Jerusalem.

EVALUATION OF THE EFFECT OF D-AMPHETAMINE SULFATE IN TREATMENT OF OBESITY IN CHILDREN. HANS-OLOF MISSBERG and A. RUNE FRISK, Acta paediat. **39**:243 (No. 3) 1950.

In a group of 167 unselected cases of obese children, besides food restrictions, every other child was given *d*-amphetamine sulfate, 2.5 mg. three times daily; children not given the amphetamine were given placebo tablets of the same appearance and with the same method of dosage. The weight loss was greater (statistically significant) in the *d*-amphetamine group than in the control group. *d*-Amphetamine sulfate is a potent aid in the treatment of obesity in children, as it is known to be in adults.

FROM THE AUTHORS' SUMMARY.

Vitamins; Avitaminoses

STABILITY OF ASCORBIC ACID IN MILK AT DIFFERENT STAGES OF PROCESSING. ARTHUR D. HOLMES, J. Pediat. **40**:91 (Jan.) 1952.

As it leaves the cow, milk may contain as much as 22.2 mg. of ascorbic acid per liter. Most of this is lost during processing, distribution, and storage. Infants need other sources of ascorbic acid.

MACDONALD, Pittsburgh.

RELATIONS OF THE ACTION OF VITAMIN E AND THAT OF THE ANTERIOR HYPOPHYSIS IN THE EVOLUTION OF GROWTH IN THE CHILD. GIOVANNI BOI, Ann. ital. pediat. **5**:24 (Feb.) 1952.

After observations regarding the similarity of the effect of vitamin E and the anterior pituitary hormone in reference to lactation (in quality and quantity) and to the development of gonads, the author reports the results of observations on some metabolic aspects; upon glycemia and glycosuria; on hematic and urinary creatinic bodies, and on chloruremia and chloruria, both in normal and in sick subjects (diabetes mellitus, diabetes insipidus, muscular dystrophy, pseudo-muscular hypertrophy).

In both the positive and the negative aspects of the observations the actions of the two elements, considered in their results, appeared in general to be closely similar.

Very interesting are the modifications of creatinic bodies in progressive muscular dystrophy.

FROM THE AUTHOR'S SUMMARY.

Hygiene; Growth and Nutrition; Public Health

SPEECH CORRECTION REFERRAL IDEAS OF CLERGYMEN. C. J. KIRKPATRICK, J. Speech & Hearing Disorders **17**:305 (Sept.) 1952.

Ninety Pittsburgh clergymen were interviewed using a questionnaire containing a description of six hypothetical problem cases, in three of which there were speech difficulties, and a series of questions asking whether or not they knew people with speech problems. A majority of the clergymen knew people with speech defects. Most had practical referral ideas but had limited information about speech correction facilities. Less than one-fourth of the clergymen contacted could have given direct referral suggestions as to institutions or individual speech correctionists with membership in the American Speech and Hearing Association.

PALMER, Wichita, Kansas.

A STATISTICAL REPORT ON FREQUENCY AND DURATION OF BREAST FEEDING AMONG INFANTS OF LOW SOCIOECONOMIC LEVEL. CARLOS A. BAUZA and ANTONIO STABILE, Arch. pediat. Uruguay **23**:171 (March) 1952.

The records of 3,977 infants aged less than 12 months were studied and the following tables were made: age and type of feeding on admission, duration of breast feeding, and cumulative percentage of duration of breast feeding. Mean duration of breast feeding ranged between 4.2 months (state well-baby clinic) and 2.1 months (infants' ward in a children's hospital).

FROM THE AUTHORS' SUMMARY.

SOME BIOLOGICAL NORMAL VARIATIONS OF CHILDREN IN HIGH ALTITUDE. CARLOS FERRUFINO and CECILIO ABELA, Bol. Soc. boliviana pediat. **3**:No. 2 (Dec.) 1949.

The absence of any anthropometric tables for Bolivian children, the impossibility of using those of other countries and the apparent difference which exists between children in high altitudes and other geographic areas led these investigators to make a study covering two and one-half years.

Since it was to discover the difference, if any, between the children of high altitudes and those at other geographical areas, the children were divided into those of European ancestry and those of mixed or indigenous population.

The plan was approved by the Cooperative Service of the Inter-American Health Department and the Bolivian Pediatric Society.

The purpose was to give some guide to pediatricians wishing to evaluate (1) somatic growth and development, (2) psychomotor development, and (3) the blood picture in Bolivian children.

Only healthy children born at an altitude of 3,600 m. and under 2½ years of age were used. These children were from the Well-Baby Clinics at the Inter-American Cooperative Service of the Public Health Department in La Paz. Children of all levels of economic, racial and social status were studied.

Five kinds of data were collected: (1) general data including exact age of child, order of birth in the family, kind of food, sex, race, and age of both parents; (2) anthropometric data collected according to the methods of Stuart and Meredith, with twelve measurements taken, namely, weight, height, sitting height, cephalic circumference, thoracic circumference, suprapubic and infrapubic measurements, pelvic diameter, calf circumference, condition of subcutaneous tissue, size of fontanel and dentition; (3) blood data, including red cells, white cells, percentage of hemoglobin, corpuscular diameter, cellular volume, plasma density, blood proteins and hematocrit readings; (4) psychomotor development, determined by the important developmental advances as used by Arnold Gesell, and (5) development of speech, including three classifications, (*a*) the age of first identifiable but meaningless sounds such as "agoo"; (*b*) the age of use of 2 to 10 words, and (*c*) the age of use of more than 10 words.

Doctors who had worked together sufficiently to grade alike collected data. An expert statistician was also in consultation.

The technical details and the tables and graphs are included and are extensive. The children are classified according to sex and according to their European or indigenous American ancestry. The tables give the arithmetic mean, the standard deviation, and range of variation in all body measurements and blood study calculations.

Graphs are included comparing the results from Bolivian children with those from children of the United States and certain South American countries; namely, Peru, Argentina, Chile, and Uruguay. The authors give credit by name to groups in other countries whose material is used.

In a total of 3,189 children examined, infants showed no great variation, regardless of race, in weight and height until after the third month, when children of European ancestry took a slight lead over the children of mixed parentage or purely Indian ancestry. In both cases, the Bolivian children weighed less and were shorter than those of the same age in the United States and Uruguay.

The thoracic measurements of the Indian children were greater than of the children of European ancestry until after the eighth month of age, at which time both become approximately the same. Bolivian children showed greater thoracic measurements than Italian children and the children of other countries of lower altitudes.

The blood picture does not vary from that of children of similar age in other countries except for the corpuscular diameter, which is greater in the newborn Bolivian children. The polycythemia of high altitudes is not manifest in this study. The variations were no greater than those at sea level in the same age group.

No unusual findings were noted in the psychomotor and language areas.

The authors believe that malnutrition would account for the variations between the children of Indian and those of European ancestry.

ALLEN, Santa Fe, N. M.

BASES FOR LEGISLATION REGARDING INFANTS AND LEGAL CODE FOR MINORS. VICTOR SAMOS, MANUEL DURAN and OSCAR FRERKING, Bol. Soc. boliviana pediat. **4**:3 (March) 1950.

This is a proposal made by the delegates from Sucre to the proposed organization for the protection of minors. The discussion is divided into (1) medical, (2) legal, and (3) social aspects.

To protect the human capital of the country and assure the nonseparation of the natural "binomial of mother and child" there must be profound knowledge of social hygiene with reference to morbidity and mortality among infants and a knowledge of eugenics; there must be institutions for the assistance and protection of all phases of infant development, and maternal care should be well regulated; education of the lay public regarding infant and maternal care should be accomplished through such media as press, radio, motion pictures, and pamphlets.

Protection of the mother should include premarital and prenatal examinations to protect both mother and progeny, and organization of services the object of which is to assure the mother the best hygienic, obstetrical, economic, and moral conditions possible. All diseases transmissible to the infant should be treated during pregnancy; nutrition should be the best, and the mother should be taught the basic care of her child.

There should be economic provisions for the working mother, with adequate subsidies to relieve her of undue anxiety so that her mental and moral health would not be impaired. The authors believe that such subsidies from the government are one of the rights of the unborn, who are the wealth of the state.

There should be establishment of means of the best delivery service whether in hospital or at home, by midwives or by physicians. Well-baby conferences and clinics for those ill should be available in all provinces, especially in rural areas. A general practitioner could take care of a screening process but anomalies, etc., should be referred to specialists if and when necessary.

There should be pediatric consultants readily available, and all protective measures possible should be given, such as immunization against smallpox, diphtheria, pertussis and tetanus; protection against syphilis, tuberculosis, malaria, and rheumatic fever should be available to all children.

Day nurseries for children of working mothers, cradle homes for orphans and abandoned children, kindergartens, and children's hospitals should be available. There should also be services for the proper sanitation of schools and a department or bureau for special care for children who are deaf, retarded, blind, or crippled. These should not be mere asylums but places for education and rehabilitation.

Under 26 headings, the doctors discuss the needs for legislation and for the adoption of a code for minors, and need for special training for juvenile court judges and officials and the need to include physicians, teachers, social workers, etc., in the program for protection of youth.

The court should not be a part of an adult court, and the places of detention should not be ordinary jails, but should be special places with proper facilities for social betterment, and should include such education as will better equip the child to return to society.

ALLEN, Santa Fe, N. M.

Prematurity and Congenital Deformities

STUDY OF THE MENTAL DEVELOPMENT OF PREMATURE INFANTS. M. SCHACHTER and S. COTTE, Pédiatrie **40**:955 (No. 7) 1951.

Schachter and Cotte studied the mental level of 310 premature infants, that is, born after 6, 7, or 8 months' gestation. The following conclusions were drawn: (1) One third of the premature infants were of normal weight (over 2,500 gm.); (2) one third were seriously mentally retarded, and (3) of those over 2,500 gm., 32% were mentally retarded, so that no significant correlation between birth weight and mental level could be established.

Alcoholism of parents or grandparents was found in about one third of the mentally retarded infants and obstetric traumas in about one fifth.

GLASER, Jerusalem.

ACROCEPHALOSYNDACTYLIA. E. MARTISCHNIG and O. THALHAMMER, Helvet. paediat. acta **7**:257 (June) 1952.

A case of pronounced acrocephalosyndactylia is described at length, and two other cases are briefly mentioned. In these cases there was no evidence of hereditary transmission, nor was an exogenous cause clearly demonstrable. On the basis of his own material and a review of cases described in the literature, the authors show that the symptom complex of acrocephalosyndactylia can be traced to a disturbance in the ninth week of embryonic life.

FROM THE AUTHORS' SUMMARY.

THE SYNDROME OF EHLERS-DANLOS. A. TENCONI, Minerva pediat. **4**:414 (May 31) 1952.

Observations were made on a boy with the Ehlers-Danlos syndrome over a period of three years. The outstanding findings were syndactylism and intracutaneous scarring. Hyperelasticity of the skin was not present. There was a family history of a similar case and of three cases of syndactylism. The inherent pathology is to be placed in the chemistry of the collagen of the skin. Therapy in this case is being directed toward correcting the collagen defect by enzymes, hormones and vitamins.

HIGGINS, Boston.

DEFORMING OSTEOCHONDRODYSTROPHY (MORQUIO'S DISEASE). WERNER BUSTAMANTE and ARMANDO DOBERTI, Rev. chilena pediat. **23**:185 (May) 1952.

The authors present two cases of osteochondrodystrophia deformans, or Morquio's disease, in two brothers, one 9 years old and the other 17 months old. The clinical and roentgenographic features of the condition are described.

FROM THE AUTHORS' SUMMARY.

EMBRYOPATHIES, WITH SPECIAL CONSIDERATION OF TOXOPLASMOSIS. O. THALHAMMER, Wien. klin. Wchnschr. **64**:323 (May 2) 1952.

Selecting cases from the literature, the author shows that embryopathies are most numerous when infection of the mother with rubeola falls within the first and second month of pregnancy. Attention is called to the possibility of an embryopathy when the infection occurs in the months before conception. The author cites his observation that epidemic hepatitis two months before conception resulted in an embryopathy. It is emphasized that other infections may be responsible, particularly measles, chickenpox, mumps, and poliomyelitis, while infectious mononucleosis, herpes zoster, influenza, and infectious hepatitis have also been reported as causes for embryopathy. The distribution of lesions in various organ systems is discussed with reference to the time of the mother's infection. The mothers need not become manifestly ill; their infection or exposure to infection may be sufficient. In the case of toxoplasmosis the protozoan is transmitted from healthy latently infected mothers to the fetus. When this occurs early in pregnancy the fetus dies; when later, the birth of a postencephalic child results.

AMBERG, Rochester, Minn.

REPORTS ON PFAUNDLER-HURLER'S DISEASE. H. R. WIEDEMANN, Ztschr. Kinderh. **70**:81 (No. 1) 1951.

A very detailed report is given of three cases of this rare condition, with roentgenographic reproductions and extensive laboratory investigations including cerebral puncture. This is followed by a discussion of the entire entity with reference to 57 articles from the literature.

GLASER, Jerusalem.

FETAL HEMOGLOBIN IN PREMATURE INFANTS. H. ZEISEL, Ztschr. Kinderh. **70**:190 (No. 2) 1951.

On the basis of resistance determinations of hemoglobin against sodium hydroxide, determined spectroscopically, the amount of fetal hemoglobin and adult hemoglobin in full term infants and in premature infants was determined. During the first three months of life there is a marked increase of adult hemoglobin and decrease in fetal hemoglobin in both groups, but whereas full term infants have practically only adult hemoglobin by the fourth to fifth month, premature infants reach this stage only at seven to eight months.

GLASER, Jerusalem.

HEREDITARY CONGENITAL ANOMALIES OF THE SKELETON (DISLOCATION OF THE CAPITULUM RADII: DISLOCATION, HYPOPLASIA, OR ABSENCE OF THE PATELLA) AND OF THE NAILS. L. S. WILDERVANCK, Nederl. tijdschr. geneesk. **93**:2576 (July 30) 1949.

Wildervanck gives a description of a family of 53 members in 5 generations, 22 of whom show the following anomalies in different combinations:

Disturbances in the elbow, wrist, and finger joints; dislocation backwards of the capitulum radii; hypoplasia of the capitulum humeri, and exostosis of the processus coronoideus ulnae. The processus styloideus radii is overdeveloped, and there may be a flexion of the fingers, especially of the little fingers.

Disorders of the knee: hypoplasia or absence of the patella; dislocation of the patella to the side (the latter is most likely caused by an underdevelopment of the musculus vastus medialis), and malformation of the condyli and of the tuberositas tibiae.

Malformations of the nails: absence or dystrophy of the nails; this is worst in the nails of the thumbs and least in those of the little fingers. This also applies to another anomaly, namely, lunulae which are very great and triangular.

All these anomalies are irregularly dominant. They are probably caused by three incompletely linked genes.

VAN CREVELD, Amsterdam, Netherlands.

Newborn

LATENT HEMOLYTIC DISEASE OF THE NEWBORN INFANT. JOSEPH GRUNDORFER, J. Pediat. **40**:172 (Feb.) 1952.

The Rh-negative mother may show a rising antibody titer throughout pregnancy and still produce an apparently healthy infant. The sensitized infant shows a positive Coombs test, more significant than the maternal antibody titer. In some infants who appear to be normal at birth, as illustrated in this report, anemia may develop in the second or third weeks. There is still great lack of knowledge as to the exact effect of the maternal antibody on the infant's vital organs. Factors other than hemolysis may play a causative role in mental deficiency due to Rh incompatibility.

MACDONALD, Pittsburgh.

MELENA IN THE NEWBORN INFANT. IRVING H. ROZENFELD and JAMES R. MCGRATH, J. Pediat. **40**:180 (Feb.) 1952.

Although, as in a case reported here, the newborn infant may have tarry stools because of blood ingested from a cracked nipple, no previous data concerning the amount of ingested blood that will produce melena in the newborn is available. The authors conclude, as a result of their experiments, that as little as 2.6 cc. of ingested blood will produce tarry stools within 7 to 17 hours in the newborn infant.

MACDONALD, Pittsburgh.

RESULTS OF ADMINISTRATION OF VITAMIN B_{12} TO NEWBORN INFANTS. ROBERT F. CHINNOCK and HARVEY W. ROSENBERG, J. Pediat. **40**:182 (Feb.) 1952.

According to Chinnock and Rosenberg, supplemental vitamin B_{12} has no effect on the growth of newborn infants.

MACDONALD, Pittsburgh.

STUDY OF WEIGHT-LENGTH IN THE NEWBORN. CARLOS BAZAN, Rev. peruana pediat. **21**:27 (April-June) 1950.

The total number of newborn children from the middle class studied was 1,010, of whom 513 were boys and 497 were girls. The average weight of 433 boys was 3,587 gm.; that of 401 girls was 3,482 gm. These weights are higher than the weights of children from the lower class, which is being studied by other national investigators. The average height, in the same number of children, was 51.5 cm. for boys and 51.0 cm. for girls. The height was slightly greater than that of infants born in the common maternity hospitals.

The number of normal deliveries was 885 (88.5%); the number of dystocial deliveries was 85 (8.5%), and the number of surgical deliveries was 30 (3.0%). There were three pairs of twins and one set of triplets (0.9%).

Of 1,010 newborn children, 42 (22 boys and 20 girls) were premature (4.2%); 15 of these died between 4 and 48 hours after birth, a mortality rate of 35.7%. Of the 15 who died 12 (80%) weighed from 1,000 to 2,000 gm., and 3 (20%) from 2,000 to 2,500 gm.

Thirty-seven (3.7%) were immature infants weighing between 2,500 and 2,800 gm.

Among the 1,010 newborn children, 24 (2.4%) were stillborn; 12 of the stillborn were macerated, and the premature predominated. Eight newborn infants of normal weight died in the first few days after their birth (0.8%).

The percentage of stillborn infants (2.4%) in this study was very similar to that in any other clinic; the maternity hospitals which care for children from the poor class had a percentage of stillborn babies varying from 3.20 to 4.70.

FROM THE AUTHOR'S SUMMARY.

FAMILIAL JAUNDICE OF THE NEWBORN ASSOCIATED WITH HEPATOSTEATOSIS. A. HOLZEL and N. SHER, Arch. Dis. Childhood **27**:37 (Feb.) 1952.

Two cases of jaundice present at birth occurring in the same family were reported.

Jaundice was associated with and assumed to be due to marked hepatosteatosis which developed in utero.

Clinically the picture was that of jaundice at birth with gross liver enlargement, apathy, listlessness, and anorexia leading to coma and death.

The chemical analysis of liver tissue showed an increase in the total fat, particularly of the neutral fat fractions (76%), with reduction in the phospholipids and the cholesterol.

The relative literature is discussed, and no previous reports of jaundice at birth due to fatty liver are traced.

KELLY, Milwaukee.

EXSANGUINATION TRANSFUSION BY SUPRAUMBILICAL CUTDOWN IN HEMOLYTIC DISEASE OF THE NEWBORN INFANT. JEAN GUINAND-DONIOL and FRANCOIS THELIN, Pédiatrie **38**:417 (No. 5) 1949.

The method of introduction of the diamond catheter in the umbilical vein by cutting down above the umbilicus is described, and it is pointed out that this vein remains open several weeks after birth.

GLASER, Jerusalem.

NEONATAL SEQUELAE OF MODERN METHODS OF OBSTETRICAL DELIVERY. J. CHOSSON, Pédiatrie **40**:251 (No. 2) 1951.

An analysis of the results of the various newer methods employed in "guided delivery," such as analgesics, anesthetics, and oxytocic drugs leads the author to the conclusion that in the hands of the specialist these methods, when correctly employed and properly indicated, do not constitute a greater danger to the child than the spontaneous delivery and may offer advantages in shortening the time of labor.

GLASER, Jerusalem.

MENINGITIS OF THE NEWBORN DUE TO COLON BACILLUS: TWO INFANTS SUCCESSFULLY TREATED WITH STREPTOMYCIN AND AUREOMYCIN. P. BEYER, J. M. MANTZ, and MLLE. LOCHNER, Pédiatrie **40**:823, (No. 6) 1951.

Two infants, 7 and 9 days old, with purulent spinal fluid due to colon bacillus infection were treated with streptomycin administered intrathecally and intramuscularly and aureomycin hydrochloride administered orally. Both infants recovered.

GLASER, Jerusalem.

TWENTY CASES OF HEMOLYTIC DISEASE OF THE NEWBORN. SALVATORE FOLENA and ANNA STEFANINI, Arch. ital. pediat. e puericolt. **14**:3 (No. 1) 1950.

Twenty cases of hemolytic disease of the newborn were studied. In the greatest number of cases isoimmunization was caused by Rh incompatibility. The patients observed at birth were treated with transfusions of Rh-negative blood, and patients having icterus gravis with transfusions after blood letting. All infants so treated survived without noteworthy consequence.

Intra-marrow transfusions were usually performed in the iliac bone. In the greatest number of cases direct transfusion was performed with no disturbance. Two twin pregnancies in which twins were affected by a different form of hemolytic disease (icterus gravis and anemia) were of particular interest. In a third twin monochorionic pregnancy (isoimmunization by Hr' incompatibility) the Hr'-positive twin was very seriously affected (kernicterus), while the other (Hr' negative) showed no disease.

Attention is called to the association, in one case, of hemolytic disease with hemorrhagic disease of the newborn and to the difficulties of differentiating clinically between some forms of the hemolytic disease (late anemic forms) and some heredosyphilitic features. About that subject it must be remembered that in a case of fetoplacental anasarca (by Rh-factor isoimmunization) with positive Coombs test on the fetal blood it was possible to demonstrate the positivity of the colloidal reactions for syphilis in the mother's and in the fetal blood. At autopsy syphilitic lesions were not found in the child, nor were spirochetes found in the liver. Histologically it has been possible to show characteristic alterations of the liver and of the spleen very different from these observed in acute erythremia (di Guglielmo's disease). The authors believe that this anatomicopathologic distinction is very clear and that a likeness between these two diseases, as some authors have tried to show, does not exist. The Coombs test enabled them to prove the existence of an isoimmunization even when the familiar condition relative to Rh factor was not the typical one. Erythroblastemia, very remarkable in the cases of anasarca, was not always present in the other forms. The globular resistance often appeared greatly decreased not only in the child but also in the mother.

FROM THE AUTHORS' SUMMARY.

ISOIMMUNIZATION AND THE RH FACTOR. C. A. LANG, Lattante **23**:193 (April) 1952.

The problems occurring in Rh isoimmunization are discussed, with particular attention being paid to statistical data and the probability of the occurrence of clinical pathology.

HIGGINS, Boston.

SOME BLOOD ANALYSES IN THE FIRST 20 HOURS OF LIFE. JUAN J. MURTAGH, CARLOS E. MARTINEZ, CASTRO VIDELA, AGUSTIN D. MARENZI, and ELSA BRAEGGER, Segundas Jornadas de la sociedad Argentina de pediatria (April) 1951, p. 164.

Between the 4th and the 24th hour of life of 65 babies, 332 analyses were performed using microtechnical methods. The results have been analyzed statistically (Mean values and standard deviations are presented in the Spanish summary.).

The normal "ionogram" of the newborn infant has been determined by computing the total base (Na, Ca, and Mg) and the following anions: CO_2, Cl, proteins, and PO_4. The difference between the cations and the anions gives us the amount of the so-called "indeterminate anion."

In 12 cases all the analyses were performed from one blood specimen. Other individual values were added to these for the purpose of increasing the series.

The results are as follows:

Total base	152.6 mEq./L.
Determinate anions	144.6 mEq./L.
Indeterminate anions	8.0 mEq./L.

The existence of a slight diminution of the total electrolytes ("hypoelectrolitemia") in the newborn has been thus established vis a vis the adult (155 mEq./L.).

The increase of the indeterminate anion reveals the presence of a metabolic acidosis.

The greater values in ketonic substances and in lactic acid, compared with the adult, are responsible for the change in the acid-base balance. The comparison of the analysis of the first 12 hours and the second 12 hours has been undertaken for the purpose of determining the existence of any "difference of significance." As regards acetone, the difference between the mean value is statistically significant. In the others it is not.

The presence of an acidosis in the first 20 hours of life indicates the advantage of early nursing and the exhibition of water and glucose.

FROM THE AUTHORS' SUMMARY.

A Case of Blood Disease in the Newborn Infant with Double Isoimmunization (Rh and ABO). Carlos A. Bauza, Amparo Amarante, and German Surraco, Arch. pediat. Uruguay **23**:383 (June) 1952.

A case of erythroblastosis fetalis due to Rh- and ABO-incompatibility is reported. No hepatic or neurological sequelae were observed on discharge.

From the Authors' Summary.

Care of the Newborn of Diabetic Mothers. W. Heck, Deutsche med. Wchnschr. **77**:1069 (Sept. 5) 1952.

The author discusses the dangers for the newborn infants of diabetic mothers and the management of the mothers and infants. With regard to the infants, 10 ml. of a 25% solution of dextrose is administered by way of the cord, and the infant is very carefully watched for 48 hours. Sugar is given by rectum or perorally if possible. The administration is guided by blood sugar determination. Regulation of temperature by regulation of external heat is used. When necessary, oxygen is supplied, and lobeline and epinephrine are kept in readiness. Intravenous administration of a 25% solution of dextrose should be used only in an emergency. Instillation of two drops of epinephrine every two hours intranasally is recommended. [The concentration of the epinephrine solution is not given.] Regular feeding is given by about the 10th day. This regimen was followed successfully in six cases. Caesarean section between the 36th and 38th week of pregnancy is regarded as the least injurious to the infant.

Amberg, Rochester, Minn.

Acute Contagious Diseases

Acute Poliomyelitis. Katherine E. Dawson; Alfred C. LaBoccetta, and Anthony S. Tornay, J. Pediat. **40**:71 (Jan.) 1952.

Twenty-eight and nine-tenths per cent of the patients sent to the Philadelphia Hospital for Contagious Diseases with a diagnosis of acute anterior poliomyelitis did not have poliomyelitis. The authors discuss those disorders that resemble poliomyelitis in its early stages and review the differential diagnosis.

Macdonald, Pittsburgh.

Aureomycin in the Treatment of Beta Hemolytic Streptococcal Infections. B. B. Breese, J. Pediat. **40**:85 (Jan.) 1952.

Although relatively small doses of aureomycin were effective against beta hemolytic Streptococcus infections, there were a number of bacteriological and clinical recurrences. Early pyogenic complications were uncommon, and late complications such as rheumatic fever and nephritis were not observed.

Macdonald, Pittsburgh.

Cardiac Complications of Pertussis. Stuart H. Walker, J. Pediat. **40**:200 (Feb.) 1952.

Major Walker uses three cases to illustrate that myocardial insufficiency may be a complication of pertussis. Signs of this complication are associated with manifestations of pulmonary interstitial emphysema, possibly the result of right ventricular stress.

Macdonald, Pittsburgh.

Some More Recent Views as to Acute Poliomyelitis. P. Schiaparelli, Minerva pediat. **4**:105 (Feb. 29) 1952.

A critical review is presented, giving recent work as to the nature of the virus, the differential diagnosis, the epidemiology, the treatment, and the possibilities as to prophylaxis in acute poliomyelitis.

Higgins, Boston.

TREATMENT OF PERTUSSIS WITH AEROSOL CONTAINING STREPTOMYCIN AND "ALEUDRIN." A. PINEROLO, Minerva pediat. **4**:139 (Feb. 29) 1952.

Fifty-one patients with whooping cough were treated with an aerosol containing 0.25 to 0.50 gm. of streptomycin in a solution of 0.2% "aleudrin" (isopropylarterenol hydrochloride) once or twice a day over a period of 5 to 18 days. Improvement or complete cure was obtained in 48 of the patients. No untoward effects of the treatment were observed.

HIGGINS, Boston.

Acute Infectious Diseases

IMPORTANCE OF EARLY DIAGNOSIS IN ACUTE RHEUMATIC FEVER. ARILD E. HANSEN, J. A. M. A. **148**:1481 (April 26) 1952.

Mild or vague pains in the region of the joints, especially those of the upper extremity, are apt to be of rheumatic origin. Rheumatic pain is often present on awakening in the morning and causes the child to limp, but the pains disappear in the evening. Fatigue pains or those associated with postural derangements occur after the child gets into bed and respond to massage. Only 20 to 30% have single rheumatic attacks; two or three attacks are usual, but some patients have 10 or more episodes. Sulfadiazine in doses of 0.25 to 0.5 gm. twice daily have resulted in only 18 recurrent attacks for 885 patient-years as compared with 140 recurrent attacks in 723 patient-years in a control group not receiving sulfonamide daily. Penicillin, besides being expensive, has not been adequately evaluated.

KIMBALL, Evanston, Ill.

THE EPIDEMIOLOGY OF ASCARIASIS AND TRICHURIASIS. H. M. JETTMAR, Bull. schweiz. Akad. med. Wissensch. **6**:391 (Oct.) 1950.

Infestation with round worms and whipworms occurs rather frequently in Styria, especially in the environs of its capital, but also within the city itself. In kindergartens of some suburbs more than 50% of the children are afflicted with ascariasis; again, in others, the whipworm prevails. While ascariasis is found relatively more rarely in northern, mountainous regions, the whipworm occurs there rather frequently in certain centers.

Putrefaction and the development of bacteria suppress the further development of Ascaris eggs, so that embryonization cannot occur in the urine and only seldom occurs in the stool solids.

If stool which contains eggs is spread out on the earth, the eggs embryonize with the advent of sufficient oxygen and moisture, and a continually higher temperature; however, they are soon carried away from the spot where they were deposited through earthworms and various arthropods, mainly flies.

Details about the biology, the thermoresistance and chemoresistance, as well as the optimal conditions for the culture of worm eggs are discussed; in this connection their extraordinary sensitivity to heating above 40 C. and to an oxygen-free milieu as well as their great resistance to certain disinfectants is pointed out.

FROM THE AUTHOR'S SUMMARY.

A CONSIDERATION OF THE RATIONALE IN THE TREATMENT OF TYPHOID WITH CHLORAMPHENICOL. JULIO MOWBRAY BARBERAN, Acta pediat. españ. **8**:151 (Feb.) 1950.

Dr. Barberan gives some of the history of chloramphenicol and the type of infections in which it has been found effective, and reports two cases. He believes that there has been too little reporting of cases in which chloramphenicol has been used for children.

Since typhoid is milder in children, many have not felt its use justified, especially since it is still fairly difficult to obtain.

He reports two cases in children 19 months and 4 years old respectively. Both cases were clinically and serologically proved to be typhoid. Both patients had been treated with aminopyrine before he saw them. The initial dose in each case was 0.05 gm. per kilogram of body weight every two hours for the first 24 hours, every four hours on the second day, and on the third day every five hours. Usually only three to five days were necessary for complete recovery. Both children had renal involvement, which also disappeared with the recovery. One child had a relapse because the medication was stopped too soon.

The author says that in his judgment chloramphenicol is a specific for typhoid, acting rapidly and with lasting efficacy; that the principal effect is the lowering of temperature followed rapidly by general improvement, and that the antibiotic is very well tolerated, that, in fact, infants tolerate as large doses as adults.

For cases in which there is inability to pay there should be a bank or source supplying this need, as the economic aspect is the most serious in Spain.

ALLEN, Santa Fe, N. M.

EXPERIENCE IN THE TREATMENT OF KALA-AZAR. E. ITURRIAGA and J. GONZALES-BOTELLA, Acta pediat. españ. **8**:569-576 (May) 1950.

Kala-azar, or leishmaniasis, is not rare in Spain, but parents rarely recognize it in their children, whereas in the Levant mothers bring in their children with the diagnosis already made, and are rarely wrong. Since the chance for recovery is dependent on early treatment, diagnosis is important.

Sufficient medication with ethylstibamine is necessary for recovery. The authors give 0.15 gm. per kilogram of body weight as a total dose. At the beginning 0.5 gm. is given twice a week, then 0.10 gm., and then 0.15 gm., until 0.20 gm. has been reached. In most cases this is enough, but in two cases reported the series had to be repeated. In one case a child had been insufficiently and incorrectly treated with antimony potassium tartrate and at the end of the first series of treatment with ethylstibamine still showed many Leishman bodies in the splenic puncture.

The best way to give the ethylstibamine is intravenously. It is given slowly with mixing of the blood and drug in the syringe to allow proper dilution. In this manner 5 cc. of distilled water can be used to dissolve 20 cg. of ethylstibamine.

Supplementary treatment with liver extract, vitamins B and C, and repeated blood transfusions are necessary in severe cases and indicated in all cases.

In severe cases with evident resisance to antimony, gold salts (A) were used to stimulate the reticuloendothelial system, for it must be remembered that kala-azar is a reticuloendothelial disease.

ALLEN, Santa Fe, N. M.

SEROLOGICAL DIAGNOSIS OF KALA-AZAR. A. FERNÁNDEZ-DELGADO DE LA PEÑA and MANUEL DOMINGUEZ CARMONA, Acta pediat. españ. **8**:699 (June) 1950.

Although the diagnosis of kala-azar is possible by means of splenic and sternal punctures, it is not easily demonstrated in peripheral blood in children. Because these methods are not always easy, and the second method unreliable, the authors made a study of the deviation reaction of complement, employing methylated tuberculin as an antigen. They found it more specific and sensitive than the formol-gel test.

There were two cases reported which gave negative results for Leishmania donovani with formol-gel, but positive with methylated tuberculin. These patients were later shown to have positive spleen punctures. The authors do not feel that all other tests should be abandoned but suggest checking by more than one method.

ALLEN, Santa Fe, N. M.

PROPHYLAXIS AGAINST RHEUMATIC FEVER IN CHILDREN. FELIX GALLEGO BARCINA, Acta pediat. españ. **9**:143 (Feb.) 1951.

Under the two general headings (1) prevention of the attack and (2) prevention of cardiopathy and other sequelae, Dr. Barcina reviews rather extensively the work of writers both in English and in Spanish.

In prevention of the attack, he places emphasis on the presence of the hemolytic Streptococcus in most cases of acute rheumatic fever and the use made of sulfonamides in places where there are large numbers of children, such as schools and orphanages. Control of the patient must be close to be effective and if side effects of the drug are to be avoided.

The daily dosage of sulfadiazine is 0.5 gm. daily in children and 1 to 2 gm. daily in adults. With this dosage, a serum level of 3 mg. per 100 cc. is maintained and there is no danger of toxic symptoms.

On the subject of preventing recurrent attacks with development of cardiopathy, Barcina discusses the pros and cons of massive salicylate dosage, the removal of foci, and the care which

should be taken before and after tonsillectomies. The use of penicillin before and after operation should be obligatory. The use of roentgen therapy and radium on residual lymph tissue not amenable to surgery is also advisable.

Antistreptococcus vaccine is mentioned as being used by some clinicians. Since 1929, Dr. Gonzales Suarez has used an autogenous streptococcic vaccine.

Some doctors use aminopyrine as well as salicylates and sulfadiazine during attacks.

The author discusses the hygienic regimen for the child subject to rheumatic fever, including the need for bed rest and excellent nutrition especially as regards proteins, vitamins A and B, fruits, and vegetables. One investigator noted that the use of eggs was noticeably deficient in the cases he studied. The practical aspects of removal to a hot, dry climate are discussed.

The author believes that prevention should be a part of the public health program, and the follow-up visits by public health nurses as well as private practitioners are very valuable.

ALLEN, Santa Fe, N. M.

PURULENT TYPHOID MENINGOENCEPHALITIS. HANS-WOLGANG OCKLITZ, Monatsschr. Kinderh. **97**:1 (May) 1949.

The author describes a case of purulent typhoid meningoencephalitis in a severely ill 21-day-old boy whose spinal and ventricular fluids yielded a positive culture for typhoid bacilli. Initially, the patient responded well to penicillin (intramuscularly, intraspinally, and through the anterior fontanelle) and sulfonamides. Later enlargement of the head and cerebral atrophy, as shown by ventriculography, developed, and the patient died after three months of illness. Blood cultures were positive only early in the course of the disease; Widal tests and stool and urine cultures were always negative. Autopsy showed pyocephalus with cerebral atrophy, chronic spinal meningitis, and chronic purulent encephalitis. Peyer's patches showed changes characteristic of typhoid.

The patient's mother was found to be a carrier of typhoid bacilli, and Widal tests of her blood (1 : 100) and milk (1 : 200) were positive. Tactile transfer of organisms from the mother to the infant, placental transmission, and infected maternal milk were all considered as possible modes of infection.

The author believes that higher doses of penicillin might have prevented development of the brain abscess leading to pyocephalus and serving as the focus of the chronic infection.

HARTENSTEIN, Chicago.

Chronic Infectious Diseases

PRESENT STATUS OF CONGENITAL SYPHILIS. J.-J. MEYER-DESCHMID and J.-P. STIEGLER, Presse méd. **58**:1414 (Dec. 23) 1950.

The antisyphilis campaign in France has nearly eradicated congenital syphilis. The few cases seen are practically always the results of lack of treatment of the pregnant syphilitic. In the large series reviewed by the authors, only 1.3 women per thousand had not been treated, and this group accounted for 40 to 50% of the cases of congenital syphilis.

The authors state that a few infants showed evidence of congenital syphilis while the mothers were free from clinical or serological evidence of the disease.

DAFFINEE, Melrose, Mass.

RESISTANCE OF B. TUBERCULOSIS TO PARAAMINOSALICYLIC ACID. N. RIST and P. VERAN, Presse méd. **59**:21 (Jan. 13) 1951.

The authors take sharp exception to the concept that Mycobacterium tuberculosis does not develop a resistance to paraaminosalicylic acid. In their opinion, this resistance develops as quickly and as frequently as resistance to streptomycin. Therefore the authors suggest that the use of paraaminosalicylic acid be limited to those cases in which a collapse procedure is to be done.

The development of resistance is slower for both streptomycin and paraaminosalicylic acid when these two drugs are used together. The resistance to the latter drug is also inhibited when sulfone compounds are used in combination with it.

When the use of streptomycin and paraaminosalicylic acid is contraindicated, therapy with sulfones is advised.

DAFFINEE, Melrose, Mass.

THE MENINGEAL SIGNS IN TUBERCULOUS ALLERGY IN THE CHILD. P. PINNA, Ann. ital. pediat. **5**:16 (Feb.) 1952.

The author reports his own observations on a young girl of 10 years with meningeal manifestations and recovery.

Examining the pathogenesis, the author excludes every other meningeal form and points out such manifestations among the tuberculosis-allergic ones, following the theory already advanced by Auricchio.

FROM THE AUTHOR'S SUMMARY.

THE VALUE OF THE CADMIUM SULFATE REACTION IN CHILDHOOD TUBERCULOSIS. G. BONTEMPELLI, Lattante **20**:592 (Oct.) 1949.

The cadmium sulfate flocculation test of Wunderly and Wuhrmann was made on the blood serum of 40 patients with nontuberculous infections and of 40 patients with active tuberculosis. With 57% of the nontuberculous patients, a positive test was obtained, but the test remained positive for only 17% during convalescence, most of the tests being only weakly positive. Patients with pneumonia, typhoid, and melitensis were especially prone to have positive reactions. Of the patients with tuberculous infections, 75% had positive tests in the acute phase of the disease and 70% continued to show positive tests during convalescence. The intensity of the cadmium sulfate reaction paralleled that of a Mantoux test. This test is regarded as indicating the degree of colloid lability in the blood serum; it seems to offer some index of the severity and of the prognosis of a tuberculous infection.

HIGGINS, Boston.

SOME DATA REGARDING CHILDREN VACCINATED BY PETRAGNINI'S ANATUBERCULIN THIRTEEN YEARS PREVIOUSLY. M. VERDI, Pediatria **60**:13 (Jan.) 1952.

Records are presented as to the present status of 6 persons who as infants or children were vaccinated with Petragnini's anatuberculin thirteen years previously. Each person still had a positive tuberculin reaction, but none had any roentgenologic signs of tuberculosis.

HIGGINS, Boston.

NEW TECHNIQUE FOR FINDING THE DIFFUSION OF PARAAMINOSALICYLIC ACID IN LOCAL TREATMENT OF TUBERCULOUS MENINGITIS. F. RAGAZZINI and G. BARTOLOZZI, Riv. clin. pediat. **48**:481 (July) 1950.

The authors in an attempt to attain a more exact and practical intradermal testing technique in the local therapy of tuberculous meningitis used paraaminosalicylic acid as a substitute for streptomycin. The data obtained from the first 141 intradermal tests, executed contemporaneously with the administration of paraaminosalicylic acid and streptomycin, showed the evident parallelism existing between the two methods. The authors therefore suggest the systematic use of paraaminosalicylic acid in intradermal tests.

In fact, in 83.7% of the first 141 intradermal tests exact correspondence was evident, while in only 16.3% were the variations of the two indexes greater than 0.25%. Therefore the authors, after a critical analysis of the exactness of the method of biological dosage in cultures, were led to believe that the cause of the error was in the above-mentioned method rather than in the paraaminosalicylic acid method, the latter being a chemical and colorimetrical method affording greater precision and convenience.

FROM THE AUTHORS' SUMMARY.

ENDOPLEURAL STREPTOMYCIN TREATMENT IN PRIMARY PULMONARY TUBERCULOSIS IN CHILD. ALESSANDRO BIEBER and ROANA ROSSI, Riv. clin. pediat. **48**:513 (Aug.) 1950.

Endopleural streptomycin treatment has been tried in 72 cases of primary pulmonary tuberculosis in children from 18 months to 12 years of age (26 of them presented adenitis; 29 adenitis with paradenitis; 11, infiltrates, and 6 inflammation of the interlobar septum and adjacent tissue) with a technique of easy application, free from any trouble. The results obtained were quite satisfactory, as recovery was always obtained and in a shorter time than through the ordinary streptomycin therapy and even in cases in which streptomycin therapy was ineffective intramuscularly. Exudative forms proved most accessible to the direct action of the antibiotic.

In cases of adenitis regression was relatively slower. According to results obtained endopleural streptomycin treatment in the primary form of pulmonary tuberculosis in infants may be considered as an autonomously effective therapeutic means to be preferred to streptomycin therapy by the usual way.

FROM THE AUTHORS' SUMMARY.

VACCINATION AGAINST TUBERCULOSIS IN INFANCY. F. FAJARDO AVILES, Acta pediat. españ. 8:657 (June) 1950.

This is another discussion of the use of BCG in infants and children, except the newborn. Dr. Aviles reviews the types of vaccines used and the reports from other countries.

Children given BCG should be healthy, with negative tuberculin tests. The children after getting the vaccination should be removed from any focus of tuberculosis infection until such time as they give a positive test after being given BCG. Rigid and vigilant care should be taken to see that these children are maintained in good health.

If a child has some illness, a period of six weeks should elapse before the tuberculin test is given. If it is negative the child may have the BCG.

Children who cannot be protected from infection for the required time within their own homes should be placed in an institution for the period required to build up immunity before being subjected to infection from parents and others in their own home.

The child who has been vaccinated and after six months still shows a negative Mantoux reaction should be revaccinated.

The vaccination is effective for between five and six years. It may be repeated if conditions warrant. For revaccinations, the child should be free from any illness and should not be allergic to BCG.

BCG should be considered only as a preventive for primary infections; by prevention of the primary infection, secondary and tertiary forms should be lessened.

The persons who should be protected are (1) all persons with negative tuberculin tests, (2) children who are in contact with relatives having the disease, (3) all nurses and persons exposed to tuberculosis through their work, (4) all young men going into military service, and (5) all young persons going away to college.

ALLEN, Santa Fe, N. M.

TUBERCULOSIS IN CHILDREN OF TUBERCULOUS PARENTS. FELIX SANCHO MARTINEZ, Acta pediat. españ. 8:833 (July) 1950.

Dr. Felix Martinez, who is director of the "Cradle Home Carmen Franco," presented this paper at the Sixth International Congress of Pediatrics at Zurich.

The Cradle Home takes children from birth to 3 years of age. In this study 98 children were used, 76 who had mothers with active tuberculosis and 22 with fathers ill of this disease. All parents had been under careful observation, and the presence of the disease proved bacteriologically, by x-ray, and, in the case of those who died, by autopsy.

Although there was a predominance of males among the children whose mothers had tuberculosis, the sex distribution among those with fathers having tuberculosis is the same as noted in the general population.

In children separated from the mother immediately after birth not one case of tuberculosis has been proved. This disproves the theories of filtrable (placental) virus and the so-called latency of the bacillus.

Of the 11 children admitted to the Cradle Home after living with a mother sick of tuberculosis, 5 died of tuberculosis. (This was before the use of streptomycin.) In these children roentgenological evidence and autopsy showed both tuberculous adenitis and the pulmonary form of tuberculosis. Three of 22 children whose fathers had been ill of tuberculosis, died.

The mortality of the children with a familial history of tuberculosis was from 1.47 to 1.55% higher than of those of the same age group with no family history of tuberculosis.

The children of mothers with tuberculosis weighed less at birth than those of healthy parents. There was not this difference in children whose fathers suffered from tuberculosis. There were 9 children with marked congenital debility who were offspring of very seriously ill mothers who later died.

The nutrition of the children whose mothers had tuberculosis was definitely poorer than that of normal children, and the more serious the maternal illness the worse the child's state of nutrition.

Rickets was present in 25 to 31.4% of the children whose parents had tuberculosis, but there was no definite proof of any greater number of those with some other disease, such as anemia, than would be found from nontuberculous parents.

During the stay in the Cradle Home the children suffered perhaps slightly more mild childhood infirmities than the children of healthy parents, but the illnesses were not specific and were of the type expected in children in the first three years of life.

Dr. Martinez believes that there is a vital deficit in these children, not due to infection per se but to the unhealthy condition of the mother and in direct proportion to the gravity of the mother's illness.

ALLEN, Santa Fe, N. M.

SPONTANEOUS RECOVERY FROM DISSEMINATED TUBERCULOSIS. J. BAUZA, J. MORICE and M. NEIRA, Rev. chilena pediat. **20**:411 (Oct.) 1949.

After analyzing the different reports published of spontaneous recovery in cases of disseminations, the authors present 10 observations of their own. They comment on the different factors which might have influenced this favorable outcome, without arriving at definite conclusions. They also study the different conditions which might be confused with this picture and with which a differential diagnosis must be made.

FROM THE AUTHORS' SUMMARY.

ANATOMICAL FINDINGS IN TUBERCULOUS MENINGITIS TREATED WITH STREPTOMYCIN. ALFREDO VARGAS BAEZA, Rev. chilena pediat. **20**:431 (Oct.) 1949.

The pathologic study of 10 cases of children who died of tuberculous meningitis and who received continuous and intensive streptomycin treatment is reported.

Streptomycin prolongs the usual course of tuberculous meningitis, thus allowing the disease to follow a pattern similar to that of tuberculous inflammation in other parts of the body. This fact conditions the anatomical findings in cases of tuberculous meningitis treated with streptomycin, in which there is a general tendency to encapsulation and organization of the exudate.

The microscopic study reveals notable differences from cases of ordinary, untreated tuberculous meningitis. Fundamentally these differences are as follows: (*a*) exudate with numerous epithelioid giant-cell tubercles, (*b*) extensive calcifications, and (*c*) important fibrosis of the leptomeninges.

The prolonged evolution of this illness results in a greater quantity and extension of the exudate, which can thus be found in parts of the axis-cylinder, where usually it does not form. In close relation with this is the practically constant involvement of the spinal leptomeninges, which are found covered with an exudate, especially the posterior part, undoubtedly a result of the prolonged supine position of the patient. A permanent finding was the presence of fresh exudates alongside the old ones, which evidently is due to the clinical relapses of the disease. In no case was total healing encountered.

Hydrocephalus of different degrees is a constant complication, and its origin lies in the mechanical factors that impede the normal circulation or reabsorption of the cerebrospinal fluid. It is possible that many of the neurological symptoms, which for the clinician are difficult of interpretation, can find their explanation in the hydrocephalus and in the various degrees of constriction that the exudates exert on the brain stem.

FROM THE AUTHOR'S SUMMARY.

TUBERCULOSIS ANTIBODIES IN CEREBROSPINAL FLUID AND BLOOD SERUM DURING STREPTOMYCIN THERAPY. A. MASSENBERG and H. KUETTNER, Monatsschr. Kinderh. **97**:18 (May) 1949.

With antigen consisting of an alcohol suspension of tubercle bacillus particles, lipids, and protein, complement-fixation tests were performed on 15 patients who had tuberculous meningitis and were receiving streptomycin. Antibodies were present in the cerebrospinal fluid in all cases, and in the serum in 12 cases. The test was almost always negative in a group of patients with tuberculous meningitis.

The authors postulate that this test may be useful in the diagnosis of cases with negative bacteriological cultures, especially after streptomycin therapy has been begun. Falling titers may be considered in the prognosis and evaluation of the effect or advisability of resumption of streptomycin treatment.

HARTENSTEIN, Chicago.

RETINAL CHANGES IN TUBERCULOSIS MENINGITIS AND MILIARY TUBERCULOSIS TREATED WITH STREPTOMYCIN. P. MEZEY and I. FLESCH, Paediat. danub. **6**:121 (Sept) 1949.

The following conclusions were drawn from the examination of 100 children suffering from tuberculous meningitis or miliary tuberculosis. At the beginning of the disease a normal retina was observed in 50% of cases of pure meningitis, whereas only in 38% of the patients suffering from miliary tuberculosis were retinal changes absent. Edema or hyperemia of the papilla was associated with other ophthalmologic changes in 80% of the cases of meningitis and 40% of the cases of miliary tuberculosis. Tubercles of the chorioidea were associated with meningitis in 16%, with the miliary form in 50% of the cases, especially in young children and infants.

Twelve per cent of the patients gave a negative response to cutaneous and intracutaneous tuberculin tests. Of these patients, 6% revealed changes on the fundus; this is considered of great diagnostic importance. In older children, hydrocephalus was regularly accompanied by papilledema, while with normal papilla hydrocephalus is rare.

In 62% of the cases there was no connection between the ophthalmologic finding and the course of the disease, while in 16% they ran parallel. No connection whatever could be found between the amount of streptomycin administered and its therapeutic effect, the course of the disease, and the associated ophthalmologic changes observed.

FROM THE AUTHORS' SUMMARY.

COURSE OF TUBERCULOUS MENINGITIS IN CHILDREN. J. HOUŠTĚK and D. SRBOVÁ, Pediat. listy **5**:89 (No. 2) 1950.

The authors deal with the clinical picture, findings and therapy of tuberculous meningitis in 57 children, 23 of whom were infants up to the age of 1 year and 24 up to the age of 2 years. At least seven months after treatment was started, of the 57 children 19 still survive, 13 of whom are infants. Tuberculous meningitis appears very frequently during early infancy; complaints and symptoms tend to be lacking in the previous history. The source of infection can more frequently be detected, first of all among the members of the family. The tuberculous changes of the lungs are more pronounced in infants, it is more difficult to make the diagnosis, and the general state of the children on admission is usually very grave.

FROM THE AUTHORS' SUMMARY.

Diseases of Blood, Heart and Blood Vessels, and Spleen

THE MURAL CELLS OF CAPILLARY HEMANGIOMAS. E. J. EICHWALD, Arch. Path. **47**:78 (Jan.) 1949.

Capillary hemangiomas are usually defined as tumors composed of vascular spaces similar to those of capillaries in caliber. This definition does not imply that the walls of the spaces are identical with those of the capillary, however, and only rarely is this true. The littoral cell (endothelial cell) of the capillary is contrasted with the mural cell of the hemangioma spaces.

The author describes components of the mural elements—fibroblast-like and epithelioid cells. The walls of larger vascular units frequently display areas of bulbous thickenings. In longitudinally sectioned vascular spaces, two of these nodules may face each other. This suggests the presence of a partial or complete ring of the tissue of which they are composed. The author believes that these nodules first arise as areas of angioblastic tissue, which are later replaced by the predominantly fibrous elements from the local stroma. This focal regressive change of angioblastic tissue recapitulates the normal developmental sequence observed in the fetal vascular tree.

HAW CHAN, San Francisco.

ABSENCE OF THE PULMONARY ARTERY: A NEW CLASSIFICATION FOR PULMONARY ARTERIES OF ANOMALOUS ORIGIN; REPORT OF A CASE OF ABSENCE OF THE PULMONARY ARTERY WITH HYPERTROPHIED BRONCHIAL ARTERIES. L. J. MANHOFF and J. S. HOWE, Arch. Path. **48**:155 (Aug.) 1949.

The case is reported of a 10-month-old child found to have complete absence of the pulmonary artery. The sole source of vascular supply to the lungs consisted of hypertrophied bronchial arteries. Associated anomalies were overriding of the aorta and no trace of a ductus arteriosus. The authors present a new classification for anomalous pulmonary artery origin based on possible variations taking place in embryological development. Huntington demonstrated that the vascular pulmonary plexus arises independent of the sixth aortic arch and attains connection to the aortic bulb by uniting with the free end of the ventral root of the sixth arch. The left dorsal root of the sixth arch becomes the ductus arteriosus when its free end grows forward to meet the ventral root. Separation of the pulmonary artery (ventral root of sixth arch) from the ascending aorta (fourth arch) takes place upon the growth of a septum between the two. Thus the proposed classification is as follows: (1) persistent truncus arteriosus (incomplete development of the aortic-pulmonary septum); (2) absence of the pulmonary artery with persistent ductus arteriosus (left dorsal root of sixth arch persisting as sole arterial supply to the lungs); (3) absence of the pulmonary artery with hypertrophied bronchial arteries (absence of entire sixth arch), and (4) absence of the pulmonary artery with anomalous attachment of the pulmonary plexus to any source other than the bronchial arteries, e. g., subclavian, inferior thyroid, celiac, or superior mesenteric arteries. If the dorsal root of the sixth arch (ductus arteriosus on the left) unites with the pulmonary plexus two sets of vessels will always be found entering the hilus of the lung. This finding differentiates the anomalous pulmonary vessel from identity as a bronchial artery. If the anomalous pulmonary artery is found to arise distal to the innominate, it can never be considered a true pulmonary artery, since this position denotes the impossibility of its being originally part of the truncus arteriosus.

HAW CHAN, San Francisco.

APLASTIC ANEMIA IN SIBLINGS WITH MULTIPLE CONGENITAL ANOMALIES. (THE FANCONI TYPE.) WALTER LEVY, J. Pediat. **40**:24 (Jan.) 1952.

Walter Levy reports two male siblings with aplastic anemia of the "Fanconi type," pancytopenia associated with multiple congenital defects. In both children the aplastic anemia was first observed at the age of 6 years.

MACDONALD, Pittsburgh.

PULMONARY HYPERTENSION IN CHILDREN DUE TO CONGENITAL HEART DISEASE. FORREST H. ADAMS, J. Pediat. **40**:42 (Jan.) 1952.

Although very few instances of pulmonary hypertension have been reported in patients with patent ductus arteriosus, interatrial septal defect, or interventricular defect, Adams states that, as a matter of fact, pulmonary hypertension is not infrequent in these conditions. When it does occur it is nearly always associated with mild to moderate cardiac disability. In such cases, the pulmonary arteries may show pathological changes.

MACDONALD, Pittsburgh.

RIGHT HEART CATHETERIZATION IN PATENT DUCTUS ARTERIOSUS AND AORTIC-PULMONARY SEPTAL DEFECT. FORREST H. ADAMS, ANTONI DIEHL, and JOSEPH JORGENS, J. Pediat. **40**:49 (Jan.) 1952.

Although right heart catheterization is of questionable clinical value in patients with typical findings of patent ductus arteriosus, it is of definite value in the atypical case. In this report, several patients thought to have some other defect were found by this technique to have patent ductus arteriosus and received the benefit of surgical ligation. Right heart catheterization is also of value in establishing the diagnosis of aortic-pulmonary defect, a very rare disorder.

MACDONALD, Pittsburgh.

RELATIONSHIP OF ACTH AND CORTISONE TO SERUM LIPOPROTEINS AND ATHEROSCLEROSIS IN HUMANS. B. BLOOM and F. T. PIERCE JR., Metabolism **1**:155 (March) 1952.

The presence of giant cholesterol-containing lipoproteins, the S_f 10-20 class, had been shown to correlate well with the occurrence of atherosclerosis. Accordingly, studies were made to determine the effects of administration of corticotropin and cortisone on the concentrations of these lipoproteins in the serums of patients. Short-term courses of corticotropin and cortisone therapy and prolonged courses of cortisone therapy (up to 19 months) were not accompanied by any consistent trend in the concentrations of these lipoproteins. The authors conclude that corticotropin and cortisone are not atherogenic if the S_f 10-20 lipoproteins are used as an index.

KAPLAN, Cincinnati.

ANGIOCARDIOGRAPHIC STUDIES OF THE HUMAN FETAL CIRCULATION: A PRELIMINARY REPORT. JOHN LIND and CARL WEGELIUS, Pediatrics **4**:391 (Oct.) 1949.

Direct angiocardiography with 10 exposures per second on human fetuses in legal abortions is described. The technical solution combines the advantages of a high film quality with a possibility of assessing the dynamic course in the heart and the circulation in both the arterial and the venous circulatory system. The fetal communications in the circulation of the human fetus are distinctly visualized, as well as the various conditions in the cardiac chambers during the cardiac cycle and also the relationship of the cycle to the circulation in the efferent vessels of the fetus.

FROM THE AUTHORS' SUMMARY.

SICKLE CELL ANEMIA: A MOLECULAR DISEASE. LINUS PAULING, H. A. ITANO, S. J. SINGER, and I. C. WELLS, Science **110**:543 (Nov. 5) 1949.

The authors report a very interesting study of the electrophoretic properties of the carbonmonoxy derivatives of hemoglobins obtained from normal adults, patients with sickle cell trait, and those with sickle cell anemia. The hemoglobins of normal white and Negro adults were found to be identical in regard to the properties studied. The hemoglobins from normal persons and from patients with sickle cell anemia show a definite difference in isoelectric point and electrophoretic mobility, whereas hemoglobin from patients with sicklemia behaves as if it were a mixture of these two types, with 39% of the total behaving as does sickle cell anemia hemoglobin and 61% as normal hemoglobin.

The authors postulate that it is this difference in hemoglobins which allows affected cells to sickle rather than some difference in the cell membrane. These observations support the contention that sickle cell anemia is inherited as a single Mendelian recessive character, with the heterozygous persons (carriers) showing the sickle cell trait.

FORBES, St. Louis.

TROPICAL EOSINOPHILIA. SIXTO S. INCHAUSTEGUI C., Rev. méd. dominicana **3**:191 (July-Sept.) 1950.

The author believes that tropical eosinophilia is a true clinical entity, different from Löffler's syndrome, as the infiltration of the lung is not present, and from the increase in eosinophiles due to intestinal parasites. The etiology is often undetermined and the eosinophiles reach a maximum of 80%. The syndrome is seen only in the tropics. Four cases are reported in which there was a return to normal after treatment with arsenicals. The Hangar test, which was positive in each instance before treatment, returned to normal afterward.

SANFORD, Chicago.

PORTOGRAPHY DURING OPERATION FOR BANTI'S SYNDROME. P. SANTY, and P. MARION, Presse méd. **59**:221 (Feb. 21) 1951.

The site of obstruction to the portal circulation in the Banti syndrome may remain obscure even at operation. The authors inject iodopyracet into the splenic vein in the first stages of the operation, and from the roentgenogram can better decide the proper surgical procedure.

Among the patients described in the report, there are five children, four of them with extrahepatic stenosis of the portal system.

A boy of 13 had a large spleen and repeated gastrointestinal hemorrhages. Absence of the portal trunk was demonstrated by roentgenogram. Splenectomy and splenorenal venous anastomosis resulted in cure.

A child of 6 had a large spleen, a normal liver, and gastrointestinal hemorrhage. There was a congenital anomaly of the portal trunk, with numerous collateral veins from the splenic vein to the hepatic hilus. Splenectomy and splenorenal venous anastomosis led to a cure. One hemorrhage occurred 18 months postoperatively.

A girl of 7 with large spleen and ascites for three and one-half years, had gastrointestinal hemorrhage for one year. Kidney function on the left was absent. There was an anomaly of the portal trunk, with collateral vessels from splenic vein to hepatic hilus. Splenectomy and adrenal-splenic venous anastomosis resulted in cure.

A boy of 7, with large spleen, had gastrointestinal hemorrhage. There was an anomaly of the portal trunk at the entrance of the splenic vein. Splenectomy and splenorenal venous anastomosis led to cure.

A child of 14, with large spleen, had hematemesis. The portal vein was normal; intrahepatic veins were dilated. The diagnosis was cirrhosis. Splenectomy and splenorenal venous anastomosis led to cure.

DAFFINEE, Melrose, Mass.

HYPERELECTROLYTEMIA IN WATER DEFICIENCY, IN CASES OF OVERDOSAGE OF ELECTROLYTES, AND IN RENAL AND ENDOCRINE DISTURBANCES. A. PRADER and E. ROSSI, Helvet. paediat. acta **4**:482 (Dec.) 1949.

The authors, basing their study on 29 examples, deal with the etiology, pathogenesis, symptomatology, serum chemistry and therapy of various forms of hyperelectrolytemia (more than 155 mEq. of base per liter of serum).

A distinction is made between hyperelectrolytemia caused by water deficiency and that caused by an increase in electrolytes (overdosage of electrolytes, certain renal and endocrine diseases). In the former the extracellular and intracellular volume is reduced. In the latter the extracellular volume is increased and the intracellular volume reduced. In water deficiency, overdosage of electrolytes, and renal diseases, the hyperelectrolytemia is unstable, while in endocrine diseases it is stable. The various forms of hyperelectrolytemia show different serum-electrolyte patterns.

Particular attention is paid to hyperelectrolytemia due to water deficiency. The present state of knowledge of pathogenesis and symptomatology of salt and water deficiency is summarized. Examples are given for water deficiency in anorexia, polyuria, hyperventilation, and diarrhea. Attention is paid to the mobilization of intracellular electrolytes (potassium, phosphorus, calcium), to the possibility of osmotic inactivation of electrolytes, to the retention of sodium chloride in favor of a better excretion of nonprotein nitrogen with normalization of hyperelectrolytemia in two phases, and to clinical and chemical differences in diarrhea with hyperelectrolytemia in infants and small children. The importance of case history, clinical condition, convulsions, thirst, vomiting, sclerema and urinalysis is discussed. Water and electrolyte therapy should be given in the form of highly diluted salt solution, in order to achieve rehydration in as physiological a manner as possible without causing edema.

Hyperelectrolytemia may occur in glomerulonephritis; in such cases the intracellular and blood volume is reduced and the interstitial volume increased. Occasionally pathogenetically obscure hyperelectrolytemia is found in nephrosis.

Hyperelectrolytemia due to hormonal factors occurs not only in cases of Cushing's disease and of diabetes insipidus hyperchloremicus, but also in other endocrine disorders. It is suggested that a secondary hyperfunction of the electrolyte-regulating factor of the adrenal cortex is present in such cases.

FROM THE AUTHORS' SUMMARY.

A CASE OF HAND-SCHÜLLER-CHRISTIAN DISEASE IN AN INFANT. W. FALK and K. PRETL, Helvet. paediat. acta **5**:229 (June) 1950.

The authors describe a case of Hand-Schüller-Christian disease in a young infant and discuss their observations in the light of published cases from the world literature. The symptom complex varies quite considerably at this age from that found in the older child or the adult. The first signs of the illness rarely appear before the baby is 4 months old, and then frequently

consist of some typical cutaneous manifestations such as chronic eczema, purpura, or seborrheic dermatitis. In many instances these skin changes constitute a preliminary to some inflammatory conditions, e. g., otitis media. In addition to these skin manifestations, one nearly always encounters a swelling of the lymph nodes, usually generalized and accompanied by enlargement of the liver and the spleen. Of the well-known tetralogy of symptoms, which is scarcely ever seen in its completeness even in older children, evidence of bony changes alone may be present in the infant later in the course of the illness, and rarely exophthalmos. The blood cholesterol level is hardly ever raised. The children usually succumb to the disease in a few weeks or months, the immediate cause of death being the profound changes in the lung. On clinical and morphological grounds great similarity exists between infectious reticuloendotheliosis (Letterer-Siwe disease) and Hand-Schüller-Christian disease. In the authors' opinion pathogenesis and etiology of both conditions belong to one of the allergic reactions (infection allergy). Assuming this allergic hypothesis, one can easily understand why many investigators in recent times have often voiced the opinion that Letterer-Siwe disease is the forerunner or acute form of Hand-Schüller-Christian disease, terminating fatally in the young infant. The characteristic disorder of lipid metabolism might also be attributed to an allergic response.

FROM THE AUTHORS' SUMMARY.

AXIAL TRANSVERSE STRATIGRAPHY IN THE STUDY OF CONGENITAL HEART DEFECTS. R. BULGARELLI, Helvet. paediat. acta **7**:156 (April) 1952.

This is the first contribution of the literature to the study of congenital and acquired heart diseases by means of the axial transverse stratigraphy (tomography) according to Vallebona's method. The cases of congenital heart disease were determined by means of angiocardiography, or during operation, or post mortem. Frontal tomography was used in all cases and anterior pneumomediastinum in most cases. Characteristic pictures are described in the cases of patent ductus arteriosus and tetralogy of Fallot before and after the Blalock-Taussig operation. In Eisenmenger's complex, it has been possible to state the location and the size of the aortic arch, the degree of dextroposition of the aorta, the size of the superior vena cava, etc. In the cases of tetralogy of Fallot it was possible to determine the degree of pulmonary circulation which resulted. Enlargements of the right auricle, of the right ventricle, of the left auricle, of the left ventricle, and the associated enlargement of two, or more, of these chambers, all show characteristic pictures in axial transverse tomography.

FROM THE AUTHOR'S SUMMARY

CARDIAC DISTURBANCES IN COOLEY'S DISEASE AND IN SUBCHRONIC ERYTHROBLASTOSIS: THEIR PATHOGENETIC AND DIAGNOSTIC SIGNIFICANCE. ANTONIETTA LEONE and ADA BASCUI, Ann. ital. pediat. **4**:1 (Feb.) 1951.

The authors have examined the cardiac changes in Cooley's disease and in subchronic erythroblastosis (with subacute course and without osteopathy) in order to determine their frequency, their primary and secondary links with the disease, and the clinical and anatomicohistologic aspects, and in order to ascertain whether on the basis of these changes one can differentiate between the two types of erythroblastosis.

Conclusions cannot be drawn briefly; cardiac alterations (with cardiac hypertrophy determined roentgenologically) are far more frequent in Cooley's disease than in subchronic erythroblastosis.

The symptoms of myocardial pain (electrocardiographically determined) are more frequent in subchronic erythroblastosis but are also to be found in the chronic form.

Anatomicohistologically one can clearly see the pseudohypertrophic nature of the cardiomegaly, frequently with tiger-lily appearance, edema, swelling, homogenization and fragmentation of myocardial fibers and nuclear pyknosis.

Between the intensity of alterations, the length of erythroblastic process and the degree of erythroblastosis there is not a close link, especially for Cooley's disease.

The authors discuss the probable origin and mechanism of the changes and believe that they may not be secondary but may derive, as do the other elements of the disease, from a common source.

They believe that in subchronic erythroblastosis a constitutional metabolic disturbance may not be clear; on the contrary, in Cooley's disease it may be evident and may work so as to make pseudohypertrophic cardiomegaly much more frequent.

FROM THE AUTHORS' SUMMARY.

RESULTS OF COOMBS'S TEST IN CHRONIC ERYTHROBLASTOSIS OF COOLEY TYPE AND IN SUBCHRONIC ERYTHROBLASTOSIS. P. PINNA, Ann. ital. pediat. **4**:60 (Feb.) 1951.

After further observations on the importance of isohemolysin factors in the genesis of chronic erythroblastosis (Cooley's anemia) and subchronic erythroblastosis, the author reports the results of Coombs's test in 18 children of the first group and 22 of the second and their families.

In only three cases (two of the first group and one of the second) was this test positive, with a frequency of Rh +, within the families examined, no higher than the average of the healthy population of Cagliari.

These results confirm those already obtained with Donath and Landsteiner's test and eliminate hemolytic isoimmunizing factors in the pathogenesis of erythroblastosis.

FROM THE AUTHOR'S SUMMARY.

MEGAKARYOCYTE COUNTS IN THE HEMORRHAGIC DIATHESES. G. CALZETTI, Clin. Pediat. **32**:536 (Oct.) 1950.

Megakaryocyte counts were made of material obtained by sternal puncture, with use of Türk's solution (weak solution of iodine-potassium-iodide) and a counting chamber. Twenty-four patients having a hemorrhagic diathesis were studied. There was an increased number of megakaryocytes in 10 cases of Werlhof's disease (thrombopenic purpura), and a diminished number in 9 cases of lymphatic leukemia, 1 case of aplastic tuberculous myelosis, and 4 cases of primary marrow hypoplasia. The megakaryocyte count was found to be normal in cases of hemophilia, of Schönlein's purpura (nonthrombopenic purpura), and of vascular disease. In only those cases with an increased number of megakaryocytes will one obtain therapeutic benefit by splenectomy.

HIGGINS, Boston.

TREATMENT OF ACUTE LEUKEMIA WITH AMINOPTERIN®: PRELIMINARY REPORT. A. MARMONT, Minerva pediat. **2**:288 (June) 1950.

A 70-year-old woman with acute leukemia was treated with aminopterin® (4-aminopteroylglutamic acid). On five occasions there was normalization of the peripheral blood following medication, but no simultaneous change in the bone-marrow picture. The patient died of a circulatory collapse.

HIGGINS, Boston.

PERNICIOSIFORM ANEMIA IN AN INFANT. G. R. BURGIO, Minerva pediat. **4**:130 (Feb. 29) 1952.

Two cases of the "perniciosiform" anemia of Gerbasi are reported in children 1 year old. The author prefers the name "perniciosiform" to "megaloblastic" or "pernicious." "Megaloblastic" is merely a hematological term, while pernicious anemia in adults is different from this condition in children under 2 years, who usually have a non-histamine-resistant achylia, and who present a much better prognosis.

HIGGINS, Boston.

BLOOD FIBRINOLYSIS IN CHILDREN. E. GRASSO, Pediatria **60**:27 (Jan.) 1952.

By the method of Cervini and Ficola (*Clinica Nuova* **11**:389, 1950) the rate of fibrinolysis in the blood of 153 healthy children, 32 children with bronchopneumonia, and 7 children with acute leukemia was found to have mean values of 104, 70, and 66 hours respectively. Fibrinolysis should be considered as a factor in the hemorrhagic tendency in children with leukemia.

HIGGINS, Boston.

HEMOCHROMOCYTOMETRIC, REFRACTOMETRIC, AND LEUKOCYTIC VALUES IN HEALTHY INFANTS AFTER ONE INJECTION OF STREPTOMYCIN. ALVARO MASI, Riv. clin. pediat. **46**:425 (June) 1949.

The behavior of the hemoglobin and of the blood cells in infants after injections of streptomycin was examined

An increase in the number of red blood cells and leukocytes and also in the mean cell hemoglobin concentration was noted regularly 40 minutes after the injection.

The relation between plasma volume and blood cell volume remained constant, and consequently the concentration of hemoglobin in the red blood cell and the saturation index increased. The average volume and the index volume of the red blood cells decreased. After 120 minutes a return to the initial values was noted. The values of blood protein were constant after 40 minutes, slightly diminished after 120 minutes.

FROM THE AUTHOR'S SUMMARY.

THE INFLUENCE EXERCISED BY THE BLOOD SERUM ON THE RESISTANCE TO SAPONIN OF THE FETAL RED BLOOD CORPUSCLES AND OF THOSE OF THE PREGNANT WOMAN. AUGUSTO GENTILI and GAETANO FASCIONE, Riv. clin. pediat. **47**:485 (July) 1949.

Research on the resistance to saponin of the red blood cells of the mother and of the fetus has been studied in normal adult nonpregnant women, red blood cells of the mother and fetus suspended in their own serum and in the serum of nonpregnant women, and red blood cells of nonpregnant women suspended in the serum of the fetus and in the serum of women at term.

These researches were made to determine whether modifications of red blood cell resistance to saponin were brought about by the various serums used and whether such resistance was due not only to certain factors in the serum but also to some peculiarities of the red blood cells. The results obtained show that this resistance depends solely on the characteristics of the blood serum. The serum increases or reduces the resistance to saponin of the red blood cells to values which are the same as those of the blood from which the serum had been obtained. The authors believe that not only blood cholesterol but also other factors in the serum influence the red blood cell resistance to saponin.

FROM THE AUTHORS' SUMMARY.

GAUCHER'S DISEASE. HUMBERTO J. NOTTI and JOAQUIN J. GIUNTA, Arch. pediat. Uruguay **20**:785 (Nov.) 1949.

The case of a 5-year-old girl who had a progressive growth of the abdomen is reported. Seven months after evidence of the growth was observed she was taken to the physician, who found a great increase in size of the spleen, which continued; impairment of the general health; hypochromic and hypophasic anemia, and pain in the left side. At the age of 7 she was seen by the authors, who found slight anemia, ocher color of the skin, great increase in size of the spleen, and increase in size of the liver. In the bone marrow they found Gaucher's cells. Splenectomy was performed two years ago. The histological study revealed an idiopathic splenomegaly with large cells of Gaucher's type. The girl improved in general health, presenting only a dark coloring of the skin. Five months after splenectomy she began limping. Nine months ago the authors found the patient in good general health, the liver enlarged by 3 fingerbreadths below the costal margin, reduction of movement and pain in the left hip joint, and shortening of this limb. Roentgenologically they found signs of Legg-Calvé-Perthes disease. They conclude that in Gaucher's disease there are histological bases to explain the roentgenological changes observed in the case. They leave open the question whether occurrence of the two diseases is coincidental, or whether Gaucher's disease, present in this patient, may be an etiological factor in the osteochondritis of the left hip.

FROM THE AUTHORS' SUMMARY.

A CASE OF HAND-SCHÜLLER-CHRISTIAN DISEASE. JULIO A. BAUZA and NICOLAS CAUBARRERE, Arch. pediat. Uruguay **20**:790 (Nov.) 1949.

A 3-year-old boy, second son of healthy parents, had bronchopneumonia at the age of 14 months. A few months later seborrhea of the scalp developed, and places could be depressed in the cranium at the position of the temporoparietal regions. Because of bilateral exophthalmia,

an ophthalmologist was consulted, who sent him to a pediatrician. The child presented numerous and extensive losses of cranium bone substance in the middle of the frontal region, in the parietal on both sides, and in the occipital. The scalp was covered by yellow fatty crusts. There was protrusion of both eyeballs, which gave a special aspect to the face. Liver and spleen showed a moderate increase. There was no increase in the lymph nodes. Anemia of hypochromic type was found, with decrease in the cell value (0.48). Cholesterol of the blood measured 3.75 gm; glucose, 1.10 gm.; calcium, 12 mg., and phosphorus, 6.2 mg., per 100 cc. The roentgenological study revealed a clear image of the so-called geographic cranium. There was also loss of calcium in regions of the scapula, ischium, and diaphysis of the femur. Roentgenological treatment was begun, but a few days later bronchopulmonary complications made the situation serious, with death of the child three months later. It was impossible to perform autopsy.

FROM THE AUTHORS' SUMMARY.

EOSINOPHILES IN VARIOUS SPONTANEOUS AND EXPERIMENTAL CONDITIONS. ALFREDO U. RAMON GUERRA and NELLY TEMENSIO, Arch. pediat. Uruguay **22**:282 (April) 1951.

The authors made eosinophile counts for 121 children with various physiological or pathological conditions, spontaneous or experimental. The eosinophile counts were made according to Dunger's method.

The experimental conditions were induced with corticotropin, epinephrine, cortisone, or corticoadrenal extracts. In the case of corticotropin Thorn's test was used, with modification of dosage according to the area of body surface. There was a definite reduction in the number of eosinophiles, but the reduction was less than is found in adults. In undernourished infants there were fewer eosinophiles, but there was also a wider distribution of values.

The response to epinephrine was in general more intense, but lower values occurred in small children, particularly in the undernourished. In general the reduction in numbers was great enough so that one cannot speak, for example, of chronic adrenocortical insufficiency.

In a series of conditions of stress, such as convulsive disorders, conditions induced by intrathecal injections of streptomycin, chills, infections (especially typhoid), intestinal invagination, and splenectomy, some aspects of the evolution of the eosinophile count were studied. There was a considerable postconvulsive decrease in eosinophiles and a gradual rise on the following days.

In the "toxicosis" it was observed that lower numbers were present in the acute state of the illness, that numbers of eosinophiles increased progressively as the condition improved, and that, on the contrary, the numbers remained low in a case of fatal outcome. On the other hand, in diarrheal disturbances that do not reach the "toxic" stage the counts remain much higher. The patient is able to react positively to a dose of corticotropin.

The countershocks mechanism as evidenced in the eosinophile count seems to exclude primary cortical insufficiency as a fundamental pathogenic element in toxicosis (anhydremia, alimentary intoxication).

FROM THE AUTHORS' SUMMARY.

HYPERCHROMIC MEGALOBLASTIC ANEMIA IN A CHILD. Z. BRUNECKY, Pediat. listy **5**:32 1950.

A case is described of severe hyperchromic, megaloblastic anemia, first diagnosed when the child was 2 years 4 months old and followed for four years. Besides the anemia there were leukopenia (3,100 to 7,800 cells), with lymphocytosis and a shift to the right, thrombopenia (56,000 to 100,000 cells), increased bilirubinemia (0.9 to 1.5 mg. per 100 cc.), and increased iron content of the blood (131γ per 100 cc). The marrow was megaloblastic (7.5 to 29.5%). All antianemic treatment failed to induce an improvement, but after administration of liver extract a surprising change took place (the reticulocytes were raised from normal to over 300%), and in a short time the hemogram showed normal results. At the same time the megaloblastic marrow became completely normoblastic. A similar effect—though a less perfect one—was produced by folic acid. It was most striking that interruption of the liver treatment invariably effected a relapse of the severe megaloblastic hyperchromic anemia within 6 to 16 months. This relapse could be observed six times. All possibility of exogenous trophotoxic influence (parasites, poisons, infections) and trophophenic factors (deficiency, goat's milk, celiac disease)

was excluded. Although free hydrochloric acid could always be found in the gastric juice, the disease most probably was caused by an endogenous deficiency of Castle's intrinsic factor, which had to be continuously supplied by administration of liver extracts. The author thinks that pernicious anemia—if it appears during childhood—produces a rather different clinical picture from that occurring in adult life.

FROM THE AUTHOR'S SUMMARY.

IMPORTANCE OF HEMATOCRIT VALUES IN THE CLASSIFICATION OF THE ANEMIAS OF CHILDHOOD. J. STREJČEK, Pediat. listy **5**:86, 1950.

Wintrob's classification of anemias, based on values obtained by Philips and Van Slyke's technique, is discussed. The advantages of this method as compared with the usual hemoglobin and color index estimation by Sahli's hemoglobinometer are shown by several examples. It is stressed that this method gives sufficiently precise information on the volume and the hemoglobin concentration of the red blood cells and can very well be used instead of the time-wasting Price-Jones method.

FROM THE AUTHOR'S SUMMARY.

AMINO ACID COMPOSITION OF HUMAN HEMOGLOBIN AT DIFFERENT AGES. A. C. VAN DER LINDEN, Maandschr. kindergeneesk. **17**:231, 1949.

The fetal human hemoglobin and the adult hemoglobin differ with regard to the globin part of the molecule. At the age of about 6 months the fetal hemoglobin is replaced by the adult hemoglobin; in premature children this process takes a longer time. Till the third year only the adult hemoglobin is present, and then a second hemoglobin appears in varying quantities, reaching the maximum around the age of 7 years; then this quantity diminishes gradually but does not disappear totally.

For their growth, lactic acid bacteria need many amino acids; if one of them is lacking, there will be no growth. The growth of the bacteria depends on the added quantity of amino acid and can be determined by means of a nephelometer or by titration of the formed quantity of amino acid. If a protein hydrolysate is added to the deficient medium, the effect of the hydrolysate on growth is decreased by the quantity of the amino acid lacking. In this way the author examined the globin of five infants varying in age from 3 to 9 days of four infants 9 to 26 months old, and of eight adults 19 to 31 years old. Between the globin of the mothers and that of the other adults, no difference was found. Between children and adults, small significant differences were found in the concentrations of leucine, methionine, and glutamic acid in the globin molecule.

The second kind of adult hemoglobin, found after the third year of life, thus has a different composition from that of the first kind of adult hemoglobin. Between the fetal and adult hemoglobin significant differences were found in seven amino acids.

In connnection with this examination the author speculates on the cause of the favorable effect of addition of cow's milk to the diet on the anemia in premature babies, which arises especially after a long-continued feeding with breast milk. Breast milk and cow's milk differ in the composition of the sulfur-containing amino acids; especially the cystine content in cow's milk is smaller and the methionine content is higher than in breast milk. The author is of the opinion that addition of methionine to breast milk may have a favorable effect on the anemia of premature babies.

VAN CREVELD, Amsterdam, Netherlands.

THE SIGNIFICANCE OF THE LYSOCYTHIN THEORY FOR INTRAVASCULAR HEMOLYSIS. S. I. DE VRIES, Nederl. tijdschr. geneesk. **94**:2461 (Aug. 26) 1950.

The lysocythin content of the blood serum was studied in 23 patients with abnormalities of the spleen of different etiologies and in 6 patients from whom the spleen had been removed (all of these last-mentioned suffered from congenital hemolytic jaundice). In 65% of these patients the lysocythin content of the serum was subnormal or absent.

A study was also made of 53 pregnant women, 31 of whom were sensitized by Rh antagonism. The majority of the women had subnormal values for lysocythin in the blood serum, but there was no difference between the sensitized and nonsensitized women. Practically always the lysocythin content in the children of these mothers was low, and there was no difference between the two groups of children.

The conclusion is that there is much doubt about the correctness of the lysocythin theory as an explanation of the process of intravascular hemolysis.

VAN CREVELD, Amsterdam, Netherlands.

INTRAVITAL STUDY OF THE BONE MARROW AS A HEMATOLOGICAL METHOD OF INVESTIGATION. S. I. DE VRIES, Nederl. tijdschr. verlosk. en gynaec. **95**:1421 (May 12) 1951.

A survey is given on the development of intravital bone-marrow examination and on its clinical value.

VAN CREVELD, Amsterdam, Netherlands.

Diseases of Nose, Throat, and Ear

THE QUESTION OF OCCULT MASTOIDITIS IN INFANTS AND POSSIBLE CURE WITH PENICILLIN. KURT NITSCH, Monatsschr. Kinderh. **97**:449-452 (Jan.) 1950.

In infants severely ill with nutritional disturbances, generalized toxicity, vomiting, fever, foul stools and the findings of mild thickening, injection or retraction of the tympanic membranes mastoidectomy should be performed if other treatment, including three to four days of penicillin therapy, proves unsuccessful. These infants usually come from poor, unhygienic homes, but may be occasionally quite well nourished. Of 42 patients with what the authors called otitis with occult retrotympanic suppuration, 35 underwent surgery and 21 were cured.

HARTENSTEIN, Madison, Wis.

Diseases of Lungs, Pleura and Mediastinum

ACUTE ATYPICAL PNEUMONIA. M. JEUNE and P. GALY, Pédiatrie **39**:43 (No. 1) 1950.

Acute atypical pneumonia is usually an interstitial pneumonia with bronchiolitis, showing few clinical signs, with insidious onset, and with a mild course. It is sulfonamide- and penicillin-resistant. Roentgenologically the involvement extends from the hilus and polymorphic shadows may be seen. Etiologically atypical pneumonia may be of Rickettsial origin, or caused by a known virus such as measles or smallpox, or of unknown origin, probably caused by an unidentified virus. The diagnosis can then be made only by exclusion and should not be made unless the cold agglutination reaction is positive.

GLASER, Jerusalem.

LOBECTOMY FOR OBSTRUCTIVE EMPHYSEMA IN AN INFANT. P. GIRAUD, R. BERNARD, COIGNET, H. METRAS, and D. HARTUNG, Presse méd. **58**:1483 (Dec. 25) 1950.

An infant of 3 months, prematurely born, had failed to gain because of an intractable bronchopneumonic infection.

Roentgenograms showed the presence of a large cystic air-filled mass in the upper lobe of the left lung. This interfered with respiration and alimentation to a point where surgical intervention seemed the only solution of the problem.

After removal of the upper lobe of the left lung, recovery was rapid, and the infant seemed normal at 15 months. No evidence of tumor or inflammatory changes was discovered in the microscopic examination of the resected lung, and the authors conclude that the origin must have been obstructive emphysema.

DAFFINEE, Melrose, Mass.

Diseases of Gastrointestinal Tract, Liver, and Peritoneum

MANAGEMENT OF FOREIGN BODIES IN ALIMENTARY TRACT. THOMAS D. GREKIN and MERLE M. MUSSELMAN, Ann. Surg. **135**:528 (April) 1952.

This study extends over a period of 11 years, from 1939 to 1950, and includes 59 patients who had swallowed foreign bodies of various types. One-fourth of the patients were psychotic. Of the nonpsychotic patients 60 per cent were under 5 years of age.

In later years the authors have adopted a more conservative plan of management of gastrointestinal foreign bodies. They advise that all patients who have swallowed moderately large or sharp objects should be hospitalized until one is certain that no untoward complications

will occur and that progress is maintained. A general diet is satisfactory, but roughage should be avoided. Cathartics and gastric lavage are condemned.

Operative treatment is advised only if the following specific indications are present: (*a*) failure of a foreign body to progress; (*b*) presence of a foreign body unlikely to pass spontaneously; (*c*) presence of a foreign body likely to penetrate the bowel; (*d*) symptoms or signs of obstruction, mediastinitis or peritonitis, and (*e*) gastrointestinal hemorrhage. BANTIN, Omaha.

INTERRELATIONSHIP OF DISEASES OF THE LIVER AND THE BRAIN. A. B. BAKER, Arch. Path. **46**:268 (Sept.) 1948.

One of the most striking and as yet unsolved examples of associated organic diseases is the combined involvement of the liver and the brain. The author discusses a group of diseases in which such findings have been reported. Those which would interest the pediatrician because of the age group in which they are usually found include Wilson's disease and kernicterus. In addition, the author reports a case of congenital atresia of the hepatic duct in an infant showing associated cerebral damage as well. Wilson in 1912 made his masterful description of the rare familial disease which he called "progressive lenticular degeneration" and which is now known as "hepatolenticular degeneration" or "Wilson's disease." This disease is characterized by the progressive development in young adults of widespread involuntary movements (tremor or chorea), severe rigidity of the musculature, difficulty in articulation and deglutition, contractures and progressive emaciation, some emotionalism, and a slight degree of dementia. Also described is the ring of greenish-brown pigment on the undersurface of the cornea near the limbus (Kayser-Fleischer ring). The course is acute or chronic, invariably terminating fatally. Pathologically the most striking features are the bilateral, symmetrical degeneration of the lenticular and caudate nuclei, and cirrhosis of the liver. The cause of this condition is still undetermined, and the chief controversies center on the two possibilities that the condition is hereditary-endogenous, or exogenous and acquired. Kernicterus, a condition found in infants dying with neonatal jaundice, was given the name by Smorl in 1902, when he found at autopsy bilirubin crystals in the nuclear masses of the brain. The clinical picture can be characterized as occurring primarily in infants apparently normal at birth with uncomplicated labor. The jaundice usually appears before the second day and is intense. Shortly after the appearance of jaundice there occurs evidence of central nervous system involvement with variable symptoms. These may take the form of lethargy, drowsiness, restlessness, excessive crying, some rigidity or spasticity, and even convulsive movements and coma. There may be a slight rise in temperature, although the course is usually afebrile. The blood picture is characteristic. There are anemia, many erythroblasts, leucocytes, and reduction of platelets. Death may occur within a few days or weeks. In milder cases there may be survival with residual brain damage. With the discovery of the Rh factor the occurrence of kernicterus was linked with erythroblastosis fetalis. The pathological finding in kernicterus is the pigmentation of the brain, nuclear masses, especially those of the basal ganglia. There is frequent associated hepatic damage. The author reports a case of his own of a 6-day-old white girl in which at autopsy the liver histologically showed severe atrophy and destruction of liver cords. The author also reports the case of a 6½-week infant girl who died with atresia of the hepatic duct which produced a progressive cirrhosis. Widespread damage of the brain (similar to that found in hemochromatosis) accompanied the hepatic changes. The author believes that, in view of experimental studies, there are indications that when the liver is sufficiently damaged it allows the various intestinal toxins to reach the systemic circulation and thus to injure the brain. The exact nature of this endogenous toxin is unknown, but experimentally it appears to be related in some way to high protein intake. The author believes that the generalized, perivascular arrangement of the brain lesions confirms the mechanism of vascular dissemination of such a substance. It is hoped that a more careful study of cases of severe liver disease will reveal a greater incidence of associated brain damage.

HAW CHAN, San Francisco.

INTUSSUSCEPTION, FECAL IMPACTION, AND VOLVULUS PRODUCED BY INJECTION OF PILOCARPINE. CURTIS M. FLORY and MARJORIE R. ALLEN, Arch. Path. **47**:60 (Jan.) 1949.

Thirty-two intussusceptions were observed in guinea pigs after subcutaneous injection of pilocarpine hydrochloride. The disease was found to be more easily induced in young than in

old animals. The authors discuss these findings in relation to the high incidence of intussusception in children under 2 years of age in whom no intrinsic disease can be found as usually can be demonstrated in adults and older children. Cause for this increased susceptibility in the very young is poorly understood, but these and other authors have suggested poorly coordinated reflexes of the gastrointestinal tract, perhaps more commonly occurring hyperperistalsis in the young, the excessive lymphoid tissue in the terminal part of the ileum (the ileocecal and ileocolic regions being the primary regions of occurrence) and the ileocecal valve with protrusion of the valve into the cecum. The authors believe that an empty state of the gut has some significance, since intussusception in the human young is commonest at the time of inadequacy of mother's milk, weaning, and teething. In their experiments they found an increased incidence of intussusception in animals deprived of food for 24 hours after the injection of pilocarpine. They postulate that possibly food acts as a bolus and prevents intussusception by interfering with the maximal contraction or by making the reflex pattern of peristalsis more uniform.

HAW CHAN, San Francisco.

DIET AND CIRRHOSIS OF THE LIVER. PREM N. WAHI, Arch. Path. **47**:119 (Feb.) 1949.

This paper is a preliminary report on an investigation of the role played by diet in the production of cirrhosis of the liver in Indian infants and adults, a condition which is common among the vegetarian Hindus. In infants the disease is clinically familial, and the illness is usually ushered in by fever and other symptoms of acute infection. The histological examination of the cirrhotic livers suggests changes of a postnecrotic scarring leading ultimately to nodular hyperplasia rather than to portal cirrhosis. In experiments rats of different ages were fed a high-carbohydrate, low-protein, and low-fat diet (approximating the Hindu diet) to observe the effects on the livers. Results were as follows:

The infant rats died at an earlier age; their livers showed little or no fatty change, whereas the adult livers showed heavy fatty infiltration. Little fibrosis was noted in the weanlings' livers except in those which had survived the earlier days of the experiment. Both infant and adult rats showed in varying amounts parenchymal necrosis of the liver. On the basis of his work the author concludes that cirrhosis is not a necessary sequel to injury of liver cells, but that some noxious agent, like a toxin, bacterium, or virus, may act as a precipitating factor to produce it. Thus in the undernourished vegetarian infant, the absence of protective proteins plays an important part in allowing the liver to be "knocked out" by a precipitating factor such as an infection.

HAW CHAN, San Francisco.

SACCULATED MECKEL'S DIVERTICULUM INCORPORATED IN THE WALL OF THE ASCENDING COLON. M. D. BOSSE, Arch. Path. **47**:175 (Feb.) 1949.

Meckel's diverticulum, estimated to occur in approximately 2% of persons, varies greatly in size, shape, position, attachments, and structure. The case reported, occurring in a 17-year-old white boy, presented a unique structure and appearance of Meckel's diverticulum which prevented its recognition at operation. A sacculated dilated distal end of the diverticulum was so well incorporated into the wall of the ascending colon that there appeared to be a communication between the ileum and the colon. The patient died six days postoperatively of generalized peritonitis following resection of the proximal unincorporated part of the diverticulum. The sacculated distal end was discovered at autopsy.

HAW CHAN, San Francisco.

PYLORIC STENOSIS IN TWINS. B. W. POWELL and C. O. CARTER, Arch. Dis. Childhood **26**:47 (Feb.) 1951.

Four pairs of twins affected with pyloric stenosis are reported. Three were monozygotic with one of each pair affected. One was dizygotic with both twins affected.

Including these cases, there have been reported in the literature 17 monozygotic pairs of which 5 had only one twin affected, and 29 dizygotic pairs of which 3 had both affected. Figures are cited which show that the incidence of pyloric stenosis is the same in twins as in infants born singly.

KELLY, Milwaukee.

USE OF CAROB FLOUR (AROBON) IN ACUTE DIARRHEAL CONDITIONS. S. T. ACHAR, Indian J. Pediat. XVIII #71 117 (July) 1951.

The author used carob flour in three cases of acute diarrhea with gratifying results. Reference is made to reports from other parts of the world in the use of this product.

McGUIGAN, Evanston, Ill.

SOME CAUSES OF VOMITING CALLED "HABITUAL" IN INFANTS. EMILIO ROVIRALTA, Helvet. paediat. acta **4**:445 (Dec.) 1949.

Today one may no longer content oneself with the diagnosis of "habitual (cyclic) vomiting." The most frequent causes of the condition as determined by x-ray are partial gastric ectopia, folding of the greater curvature of the stomach, and chronic duodenal stenosis.

In partial gastric ectopia, that part of the stomach which is displaced into the thorax sometimes takes on the shape of an open umbrella and sometimes that of a cupola. The malformation, also called brachyesophagus or short esophagus, occurs at least as frequently as hypertrophic pyloric stenosis. In the former, however, vomiting occurs in the first few days of life. X-ray demonstration is best accomplished with 30 to 40 cc. of a fairly viscid barium suspension. Treatment consists of placing the thorax in a higher position than usual.

Folding of the greater curvature of the stomach is most usually caused by meteorism of the transverse colon. At times it is caused by the failure of the posterior mesogastrium to unite with the abdominal wall and a displacement of the pylorus to the left. The characteristic x-ray picture is usually seen only in the first few minutes after the ingestion of the contrast medium. Placing the patient on his stomach after mealtimes will overcome the vomiting.

Chronic duodenal stenosis can be recognized by the delayed emptying time of the somewhat dilated duodenum. Placing the patient on his stomach supported by a cushion will remove the symptoms.

FROM THE AUTHOR'S SUMMARY.

ULCER DISEASE IN CHILDREN WITH SPECIAL REFERENCE TO FREQUENCY. FRITZ KARLSTRÖM, Helvet. paediat. acta **4**:455 (Dec.) 1949.

Ulcerative conditions in children are comprised, on the one hand, of the type mainly observed in infants, i. e., a secondary ulcer as the result of other illnesses, and, on the other hand, of the type observed in older children, the causes of which are probably similar to those of ulcer in adults.

In the pediatric clinics of 26 Swedish hospitals, 30 cases of ulcerative conditions in older children (up to the age of 15 years) have been observed within the last five years. The actual total of Swedish children suffering from ulcerative conditions was undoubtedly considerably higher for this period than the above findings. The possibility of ulcer therefore should be borne in mind more generally in cases of children with stomach complaints.

Ulcerative conditions in children appear most frequently between the ages of 10 and 15 years. Boys are more susceptible to this condition than girls, and duodenal ulcers are more prevalent in childhood than peptic ulcers. The clinical symptomatology of this condition is generally the same in children as in adults.

The results of dietetic treatment of ulcer appear to be good. The indications for operative treatment should therefore be more seriously questioned in the case of children than in the case of adults.

FROM THE AUTHOR'S SUMMARY.

CHOLELITHIASIS IN CHILDREN. JOSEF WELLAUER, Helvet. paediat. acta **4**:462 (Dec.) 1949.

The author describes a case of cholelithiasis in a 4-year-old girl. Reports of 245 other cases of cholelithiasis in childhood are collected and analyzed, the incidence at various ages calculated, the clinical picture briefly described, the main symptoms stressed, and the likelihood of diagnosis statistically assessed. Attention is drawn to the importance of bearing in mind the possibility of cholelithiasis in all cases of severe stomach pains of no specific cause; a careful and unprejudiced examination should be made with this in mind, and with particular thought to the development of the clinical picture.

FROM THE AUTHOR'S SUMMARY.

FURTHER RESULTS IN TREATMENT OF DYSPEPSIA AND OF ENTERITIS IN INFANTS WITH PUMPKIN. M. ORLANDELLI, Clin. pediat. **33**:69 (Feb.) 1951.

Treatment with puree of pumpkin has been used at the Pediatric Clinic of Bologna in 109 cases of dyspepsia or enteritis with very satisfactory results.

HIGGINS, Boston.

FIBROCYSTIC DISEASE OF PANCREAS. A. MASI, Riv. clin. pediat. **48**:349 (June) 1950.

The author, after briefly summarizing the clinical picture concerning fibrocystic disease of the pancreas, explains the etiopathogenitic, diagnostic, and curative problem. He reports two cases which he studied personally in the pediatric hospital in Florence.

FROM THE AUTHOR'S SUMMARY.

DIAGNOSIS AND TREATMENT OF TYPICAL INTUSSUSCEPTION IN INFANT. P. PRIETO and L. ALONZO DE LA TORRE, Acta pediat. españ. **90**:635 (June) 1950.

The authors review the literature on intussusception, the diagnostic symptoms, the treatment, both surgical and by enemas, and the rarer forms.

Among the rarer forms is the invagination of a portion of the colon into another section of the colon. According to the statistics of Ladd and Gross there are only 2% of this type. One of the cases reported falls into this classification. In this case the hemorrhage was an early sign and there was no vomiting.

Another rare form (5%) is the ilioiliac or enteric form, in which there is intense pain and early vomiting, while hemorrhage is late or may not appear at all. In such cases the abdomen is tympanitic, vomiting persists, and the signs of intussusception may be obscured by the signs suggestive of obstruction.

Another rare form is the ascending or retrograde invagination. Most cases of intussusception are descending; that is, the upper segment is caught in the lower segment, but the reverse may be found in rare cases.

The authors discuss preoperative care, types of operations, and postoperative care; also the use of cyclopropane and local anesthesia in these cases.

In case 1, a child of 6 months had an ileocecal-colon type of invagination but suffered no apparent pain, vomited once only, and did not cry in an unusual way. The child was given an enema by the mother shortly after the vomiting episode, and when blood was found in the feces she called the physician. There was a mass palpable in the area of the hepatic flexure, and x-ray examination with opaque enema confirmed the diagnosis of intussusception. Surgical intervention was necessary. Even then the invagination was reduced with difficulty. There was an area which appeared discolored, but the normal color returned shortly after the reduction.

At no time did the child flex his thighs in pain or give any of the sharp agonizing cries associated with this type of intussusception. Recovery was rapid and complete.

The second case was similar in all respects except that the invagination was quickly reduced by a barium enema. Some small blood clots and mucus were passed before the enema, and when the enema was expelled a foreign body was also expelled. It was a small rubber nipple.

In the third case there were extreme pain and diarrhea with definite crises of pain, but no vomiting. The mass was in the left inferior quadrant, but blood was noted only after a digital rectal examination. Surgical intervention was necessary, and the transverse colon was invaginated to the distal portion of the descending colon. There was an uneventful recovery. The authors repeat that at no time was vomiting present.

In the fourth case there were definite crises of pain but there was no hemorrhage; the patient recovered without surgical intervention.

ALLEN, Santa Fe, N. M.

ELECTROCARDIOGRAM IN GASTROENTERITIS (TOXIC SYNDROME) OF INFANTS. J. RINGEL, Pediat. listy **5**:94 (No. 2) 1950.

The author found flattening of the T wave in all leads and its absence or inversion in the extremity leads in 35 electrocardiograms of infants with toxic diarrhea. These changes appear gradually and signify an impoverishment of the organism in important electrolytes, mainly in

potassium. He and his associates also found a prolongation of the Q-T interval and an oblique course of the ST segment, which continues smoothly into a negative T wave. A definite correlation was observed between the degree of the electrocardiographic changes and some of the signs of the toxic syndrome, especially the dehydration, which is accompanied with the loss of some of the electrolytes. The evolution of the electrocardiogram respresents, therefore, a means of checking the progress of the rehydration process.

FROM THE AUTHOR'S SUMMARY.

GRAM-NEGATIVE STAINING OF GRAM-POSITIVE INTESTINAL BACTERIA WITH REFERENCE TO EXAMINATIONS OF FECES IN INFANTS. CURT STAHL and ERIK OLSEN, Acta paediat. **39**:471 (No. 6) 1950.

The observation made by E. Olsen that dead Gram-positive intestinal bacteria of young infants stain Gram-negatively in smears of feces is confirmed by the experiments which have been carried out in the present work. A combination of pepsin, pancreatin, and bile accelerated the change to Gram-negativity which occurs spontaneously in vitro in heat-killed Thermobacterium lactis, Thermobacterium intestinale and lactobacillus parabifidus. A distinct effect is observed even within the first 24 hours. A few experiments suggest that L. parabifidus is most strongly influenced by pancreatin alone, whereas the combination of enzymes and bile has the greatest effect on Tbm. lactis.

FROM THE AUTHORS' SUMMARY.

Nervous Diseases

SPONTANEOUS DEMYELINATING DISEASES OF ANIMALS: A STUDY IN COMPARATIVE PATHOLOGY. LESTER S. KING and MARJORIE C. MEEHAN, Arch. Path. **46**:567 (Dec.) 1948.

The authors define a demyelinating disease as one in which the disease process attacks primarily and essentially the white matter of the brain. Using such a criterion, one finds several important diseases of lower animals which appear to be discrete nosologic entities. In monkeys and apes a condition has been observed, of subacute to chronic course, characterized by demyelination of the brain, optic nerve tracts, and spinal cord. No infectious or transmissible agent has been demonstrated. Dietary or other environmental factors are suggested as causal, but proof is lacking. Through the work of Innes and of Bennetts, a disease of fetal and young lambs has been well defined. Demyelination and, in severe cases, softening leading to cavitation form a distinct disease entity. There is overwhelming evidence that copper deficiency of the pregnant ewe plays a role in the etiology and that the disease may be prevented by supplementary copper feedings. However, the mechanisms at work are not known. In horses a fulminating disease with destruction of white matter of the cerebral hemispheres has been studied. Etiologically it has been traced to the ingestion of moldy corn and appears to be "toxic" in nature. In moose of both Maine and Minnesota spotty demyelination has been observed with well defined glial scars appearing in the white matter of the cerebral hemispheres. The clinical syndrome is not clearcut, no transmissible agent has been found, and the cause is obscure. Encephalomyelitis of dogs and goats has been described, with variable destruction of white matter. This damage is in part caused by localization of infectious agents (Listerella in goats and distemper virus in dogs). But in some dogs no infectious agent can be traced, and it appears unquestionable that in dogs more than one mechanism is at work to cause the damage of the white matter. The importance of destruction of the white matter as a process, rather than as a disease, is stressed.

HAW CHAN, San Francisco.

CASE OF CHILD WITH LATENT PSYCHOMOTOR EPILEPSY, DIAGNOSED BY ENCEPHALOGRAMS AND TREATED BY LEVOGLUTAMIC ACID AND DIPHENYLHYDANTOIN. C. LOMBROSO, Pediatria **58**:393 (May) 1950.

An 8-year-old boy was slow in learning to talk and had shown poor social adaptation. He did not do well at school and was believed to be feebleminded. But he was found to have an intelligence quotient of 90. Electroencephalograms showed subclinical psychomotor epilepsy. Treatment was by daily doses of levoglutamic acid; diphenylhydantoin sodium was also given

in doses sufficient to render the brain waves regular. The child showed marked improvement socially and was able to return to school, although the intelligence quotient remained at about 90.

HIGGINS, Boston.

A CONTRIBUTION TO THE STUDY OF MENINGITIS DUE TO HEMOPHILUS INFLUENZAE (PFEIFFER). MANUEL DE TALASA-ZATOUR, Acta pediat. espān. **10**:54 (Jan.) 1952.

The history of meningitis due to Hemophilus influenzae is reviewed. The difficulty of laboratory diagnosis due to the morphological types and the phases of its development as elaborated by Margaret Pittman are discussed.

The course of the disease and its treatment by sulfonamides, streptomycin, and the rabbit serum of Alexander are included. The author says that treatment with sulfonamides is only partially satisfactory; with streptomycin alone or with the sulfonamides, results have been good. The author presents 10 cases of good recovery with streptomycin. He gives the minimum dosages used in the 10 cases by intramuscular and intravenous routes, and also the minimum concentration of streptomycin effective to prevent the growth of the organism on Levinthal agar after 48 hours. The latter varied in the 10 cases from 1.6 to 7.5%.

The author describes the use of the Alexander serum, but says that he has had no experience with this mode of treatment.

ALLEN, San Francisco.

THREE CASES OF SUPPURATIVE MENINGITIS WITH PETECHIAL ERUPTIONS TREATED AND CURED WITHOUT USE OF INTRASPINAL THERAPY. JOSE BONABA, JUAN GIAMPIETRO, and VICTOR CAPDEPON, Arch. pediat. Uruguay **20**:839 (Dec.) 1949.

Three cases of acute purulent meningitis are presented, probably of meningococcal origin, because of slight skin hemorrhages and simultaneous appearance in an almost epidemic form, although the causative agent could not be found in various studies. The patients were all treated by the simultaneous use of penicillin and streptomycin by intramuscular route, the intrathecal route being completely omitted. The total doses of penicillin varied between 3,325,000 units (25,000 per kilogram of body weight) and 6,640,000 units (43,000 per kilogram); those of streptomycin between 40 and 67 gm. (0.25 and 0.43 gm. per kilogram of weight). Recovery was complete in all three cases.

FROM THE AUTHORS' SUMMARY.

A CASE OF ACUTE PURULENT MENINGITIS WITH RECOVERY ON TREATMENT BY EXTRATHECAL ROUTE. JOSE BONABA, VICTOR SCOLPINI, and JUAN CARLOS ANGELILIO, Arch. pediat. Uruguay **20**:848 (Dec.) 1949.

A 4-year-old boy had acute catarrh five days and presented symptoms of acute meningitis three days before admission to the hospital. On admission meningitis was found by the examination of the cerebrospinal fluid, but the causative germ was not discovered. The patient was treated with the triple combination of penicillin, streptomycin, and sulfathiazole, with total doses of 2,400,000 units, 2,800,000 units and 48 gm., respectively, the first two being used only by the extrathecal route and the third one by the oral route. The patient showed dramatic improvement in 48 hours; there was total recovery.

FROM THE AUTHORS' SUMMARY.

INTERPRETATION OF THE ELECTRO-ENCEPHALOGRAPHIC DATA IN ENCEPHALOPATHIES IN CHILDREN. M. TURNER, Arch. pediat. Uruguay **23**:165 (March) 1952.

The author considers it useful to introduce the idea of impulses originating in the "areothalamic sector" in the interpretation of electroencephalographic findings in cerebral palsy in children. They could originate in the subcortical pole of one or two hemispheres as well as in the cortex. To illustrate this he presents a case of cerebral palsy with epilepsy and syndrome of Little in which the electroencephalogram activated by rhythmic photic stimulation shows the subcortical origin of the disturbances. Finally and principally, this prevents the possibility of contemplating surgical intervention in "cortical cicatrix" which is surely nonexistent.

FROM THE AUTHOR'S SUMMARY.

ACUTE SUBARACHNOIDAL HEMORRHAGE IN CHILDHOOD. H. PRENZEL, Kinderärztl. Praxis **18**:111 (No. 3/4) 1950.

Three cases of subarachnoidal hemorrhage in children aged 7 to 11 years were observed within four months, two occurring on the same day. The main findings were sudden severe headaches, nausea, vomiting, apathy, coma or restlessness, convulsions, collapse, neck rigidity, retinal hemorrhages, bloody cerebrospinal fluid, and xanthochromic supernatant fluid. In two cases a history of increased intracranial pressure due to prolonged downward position of the head could be obtained; the third child was found in coma.

The author believes that congenital weakness of the blood vessels is the cause of this condition and that changes in intracranial pressure and possibly meteorological influence act as trigger mechanisms.

GLASER, Jerusalem.

EPILEPSY AND LEFT-HANDEDNESS. W. HORDIJK, Nederl. tijdschr. verlosk. en gynaec. **96**:263 (Feb. 2) 1952.

In accordance with the views held by others, the author found that in "left-handed" families epilepsy occurred nearly 15 times as often as in the "right-handed" group. These correlative anomalies therefore may be of value in the early diagnosis of epilepsy.

VAN CREVELD, Amsterdam, Netherlands.

ISOLATION OF THE HERPES SIMPLEX VIRUS FROM THE BRAIN OF A PATIENT WITH ACUTE ENCEPHALITIS. H. A. E. VAN TONGEREN and J. G. Y. DE JONG, Nederl. tijdschr. verlosk. en gynaec. **96**:871 (April 12) 1952.

The herpes simplex virus was isolated from the brain in a case of fatal meningoencephalitis in a boy nearly 4 years old. The prodromata included severe headache and vomiting; later the patient showed high fever and apathy. On the sixth day of illness the boy lapsed into coma and suffered convulsions, tremors, spasticity of the legs, disturbed reflexes, and gastrointestinal disturbance. Shortly before death papilledema with hemorrhages of the fundus oculi was seen.

Cross-immunization tests carried out between the freshly isolated virus strain and an established strain of herpes virus showed them to be immunologically identical.

VAN CREVELD, Amsterdam, Netherlands.

COMMOTIO CEREBRI. A. VERJAAL, Nederl. tijdschr. verlosk. en gynaec. **96**:877 (April 12) 1952.

The author surveys recent conceptions on the nature of concussion of the brain; he insists on sharper diagnosis and more precisely planned therapy. Bed rest should last from a few days to some weeks. No advantage may be expected from strict horizontal position. If the patient still has complaints some weeks after a concussion three possibilities must be considered: (*a*) complication by an organic defect as a consequence of a contusion or a hemorrhage; (*b*) existence of a disease which has no relation to the trauma, or (*c*) a post-traumatic neurosis.

VAN CREVELD, Amsterdam, Netherlands.

Psychology and Psychiatry

A PEDIATRICIAN LOOKS AT PSYCHIATRY. LANGLEY PORTER, J. Pediatrics **38**:275-284 (March) 1951.

Dr. Porter has traced the progress of medicine over the last half-century. He outlines the development of modern psychiatry through Freud and Jung and Adler. With due credit to the others, he thinks that Adler's concept of the psyche is most useful to the clinical pediatrist. The idea that unsatisfied self-assertion is the main cause of neuroses appeals to the pediatrist. For Adler, what he calls the "style of life" is established in the first five or six years of childhood. Thereafter, the child's reactions to various emotional stimuli follow an acquired pattern. The family circle is the determining influence on this pattern. It is the duty of the pediatrist to help the family to avoid the obvious errors and injustices too often visited on children and to assert the unique individuality of the child and his right to freedom within the limits

of parental responsibilities. It is impossible to detach the child's behavior from the family situation. The postulates fundamental to sound family relationship for children are courtesy, certainty, and consistency. Families that follow these three C's rarely have problem children.

MACDONALD, Pittsburgh.

TREATMENT OF MENTALLY DEFICIENT CHILDREN WITH GLUTAMIC ACID AND WITH FOODS RICH IN GLUTEN. F. FORTUNATO and B. CANELLA, Clin. pediat. **33**:77 (Feb) 1951.

Ten mentally retarded children were given 8 to 10 gm. of levoglutamic acid daily for periods of 6 to 11 months. Another group of five mentally retarded children were given food rich in gluten. Still another group of 13 served as controls. Both the children receiving glutamic acid and those on a high gluten diet showed encouraging improvement in their intellectual development; the mean change in their intelligence quotients was from 0.567 to 0.590. The control group, on the other hand, showed a drop in their intelligence quotients, the change being —1.5. As satisfactory improvement was noted with the children receiving the gluten-rich diets as with those receiving the levoglutamic acid.

HIGGINS, Boston.

PHENYLKETONURIC MENTAL DEFICIENCY: FÖLLING'S DISEASE. F. LOTTI, Clin. pediat. **33**:110 (Feb.) 1951.

Three cases of Fölling's disease in siblings (one boy and two girls) are reported. This condition is characterized by mental retardation, albinism, blue sclerae, and the appearance of phenylpyruvic acid in the urine. The family history revealed no other cases. The disease seems to be caused by a defect in the metabolism of phenylaminopropionic acid and to be benefited by attempts to correct this deficit.

HIGGINS, Boston.

STUDY OF A CASE OF JUVENILE DELINQUENCY WITH AGGRESSIVE-NEUROTIC ATTITUDE. M. SCHACHTER and S. COTTE, Riv. clin. pediat. **50**:115 (Feb.) 1952.

The authors describe the case of a young boy of 14 years 9 months, seen because of his fugues and money stealing. The detailed study of this case shows the role played by a concurrence of constitutional, heredopathologic, mesologic and psychosocial factors. The analysis of this deviated personality is made in the light of the current tests of the projective psychology (Rorschach, T. A. T. van Lennep). In conclusion they give some medicosocial considerations concerning the handling of such subjects.

FROM THE AUTHORS' SUMMARY.

PSYCHOLOGY AND PEDAGOGY OF ATTITUDE OF DEFIANCE IN EARLY CHILDHOOD. H. STUTTE, Kinderärztl. Praxis **18**:17 (Jan.-Feb.) 1950.

A discrepancy in the development of various phases of the child's character between 2 and 4 years of age leads to its normal and natural opposition to parental authority. Drive toward independence and desire for love and mothering create a conflict in the personality of the child at this period of development which is difficult to understand and still more difficult to tolerate. This leads in many cases to the attempt of the parents to "break the opposition," which is quite in contrast to what they should do if they desire to develop the child's personality in normal channels. Patience, understanding, and diplomatic guidance should be the motives of the parents' attitude toward the child during this period of emotional lability and irritability.

The case of a child is described in whom the phase of defiance developed at the age of 2½ years and continued in pronounced form for almost two years. The situation found an accidental beneficial solution when a member of the family composed a humorous poem about the child's behavior at various occasions during the day. The frequent quotation of this poem caused laughter and a pleasant relaxed solution of the tense atmosphere around the child. The child's negativistic attitude changed within a period of two to three months, and a pleasant parent-child relationship developed.

GLASER, Jerusalem.

RELATION BETWEEN THE MONTH OF BIRTH AND SCHIZOPHRENIA AND MANIC-DEPRESSIVE PSYCHOSES. W. J. J. DE SAUVAGE NILTING, Nederl. tijdschr. verlosk. en gynaec. **95**:3855 (Dec. 15) 1951.

As in a previous study in 1934, the question was investigated whether a certain correlation existed between schizophrenia and analogous disease and the month of birth. The same relationship was found, a distinct rise of the curve during the winter months. This connection was not as evident for manic-depressive psychosis. Further research is necessary. In order to explain this surprising phenomenon, it is suggested that the deficiency of certain vitamins in winter may have a harmful influence on the fetus, which manifests itself in the increased tendency toward one of the diseases mentioned.

VAN CREVELD, Amsterdam, Netherlands.

CHILDREN WHO DO NOT SPEAK. F. GREWEL and M. L. WATERMAN, Nederl. tijdschr. verlosk. en gynaec. **96**:74 (Jan. 12) 1952.

On the basis of four groups, namely, deafness, feeblemindedness, neurologic diseases and peripheral-mechanical diseases, and other diseases, the authors review the causes of inhibition of the development of speech and of the use of language.

The cooperation of the pediatrician and of the general practitioner in an early recognition of these disorders is required.

VAN CREVELD, Amsterdam, Netherlands.

Diseases of Genitourinary Tract

CONGENITAL MEGALOURETER AND HYDROURETER. G. E. IRVIN and JOHN E. KRAUS, Arch. Path. **45**:752 (June) 1948.

Nonobstructive enlargements of the ureter can be divided into (1) those associated with neurological lesions such as spina bifida and (2) those for which no organic cause is found. It is the latter with which this paper is concerned. The term megaloureter in most textbooks of urology usually defines a huge, dilated, atonic ureter without evidence of obstruction at its opening. Megaloureter has been recognized in fetuses, newborn infants, and children for many years, although it is an uncommon cause of enlargement of the urinary tract. It is found frequently associated with other developmental defects, particularly those of the gastrointestinal tract. Megaloureter may be unilateral or bilateral. If unilateral the condition usually causes no symptoms clinically. If bilateral, trouble begins early as a result of urinary stasis and subsequent infection. Suggested theories of pathogenesis include (1) previously existing obstruction which has disappeared by the time of examination; (2) developmental malformation; (3) ureterovesical achalasia, and (less plausible theories) (4) thiamine deficiency, and (5) inflammation at ureterovesical junction. Four cases of congenitally enlarged ureters are presented in detail representing different types. The authors conclude that there will be found congenitally enlarged ureters defying classification.

HAW CHAN, San Francisco.

ANESTHESIA IN PEDIATRIC UROLOGY. STEVENS J. MARTIN, and THOMAS M. FEENEY, J. A. M. A. **148**:180 (Jan. 19) 1952.

Anesthesia in children is a specialty within a specialty; the sensitivity of the child's nervous system to various agents must be taken into consideration, and also the relatively greater need for oxygen and the relative smallness of the infant's or child's airway.

KIMBALL, Evanston, Ill.

CHRONIC AMINOACIDURIA IN GLYCOGENOSIS AND CYSTIC DISEASE. G. FANCONI and H. BICKEL, Helvet. paediat. Acta **4**:359 (Nov.) 1949.

The syndrome described in this article is made up of the following principal symptoms: (1) aminoaciduria, probably with normal amino-acid blood level; (2) dwarfism; (3) urine changes, consisting in albuminuria, polyuria, glycosuria, phosphaturia, generally alkaline urine, and greatly increased ammonia excretion; (4) blood changes, consisting in hypophosphatemia

with normal phosphatase values, hypercholesteremia, low alkali reserve, and glycolability; (5) skeletal changes, sometimes resembling rickets, sometimes merely osteoporosis (osteodystrophia pseudorachitica), and (6) thermolability ("thirst fever") and polydipsia.

This syndrome may occur in combination with other more obvious manifestations, particularly with cystine storage in the whole body, especially in the reticuloendothelial system. A case in point is described, in which the brother also died of cystinosis.

Freudenberg, Hottinger, and others consider the cystine storage to be the primary factor. The syndrome has, however, frequently been observed without any traceable cystine storage. A case showing the combination of this syndrome with glycogenosis is described, apparently for the first time.

The authors consider the scheme drawn up by Fanconi in 1946 (*Helvet. paediat. acta* **1**:201) of the pathogenesis of the cystinose as in the main correct. They regard the disturbance of the amino-acid metabolism, probably the disturbance of the desamination of the amino acids, as the primary factor in the disease.

Chronic aminoaciduria is a hereditary primary metabolic disorder. Acute aminoaciduria can, on the other hand, occur secondarily as the result of certain severe liver diseases with extensive cellular destruction. This is "true amino-acid diabetes" with high amino-acid blood level, in contrast to the "renal amino-acid diabetes" which is usual in cases of nephrotic glycosuric dwarfism without cystinosis. Paper partition chromatography, a diagnostic method recently introduced into clinical medicine, now enables one to make a qualitative and rough quantitative assessment of the aminoaciduria, the most characteristic feature of the syndrome.

FROM THE AUTHORS' SUMMARY.

PERSISTENT FAMILIAL PHOSPHATE DIABETES WITH VITAMIN-D-RESISTANT RICKETS. G. FANCONI and P. GIRARDET, Helvet. paediat. acta **7**:14 (March) 1952.

Two cases of vitamin-D-resistant rickets, in mother and son, are reported in which the main blood chemical abnormality was chronic hypophosphatemia. In spite of enormous doses of vitamin D, well tolerated by the child, an only moderate improvement of the rachitic bone changes resulted, and the hypophosphatemia remained unchanged. The serum-phosphatase level was not significantly elevated, and the blood-calcium level was normal, the latter ruling out primary hyperparathyroidism. Examination of the urine revealed nothing significant except for increased phosphate excretion, associated with a minimal excretion of calcium. The authors advance the theory that this is a case of "phosphate diabetes," probably of renal origin.

The determination of phosphate clearance yielded the enormous clearance value of 2,760 cc. per hour per 1.73 sq. m. body surface, compared with 130 and 357 cc. per hour and 1.73 sq. m. body surface in child controls. As was to be expected, the Ellsworth-Howard test gave an essentially negative result: Parahormone administration increased phosphate excretion only 17% and 28%, implying that the already minimal tubular phosphate reabsorption, caused by the disease process, could not be further blocked. The authors suspect that the approximately 30 reported cases of vitamin-D-resistant rickets in the literature are examples of phosphate diabetes, especially the hereditary cases.

Whereas in phosphate diabetes only the reabsorption of phosphate is blocked, in cases of the Debré-de-Toni-Fanconi syndrome there is also interference with glucose and amino-acid reabsorption, a fact which explains the much severer disease picture in the latter.

FROM THE AUTHORS' SUMMARY.

LIPID NEPHROSIS AND ITS TREATMENT WITH CORTICOTROPIN (ACTH). WILLY TANGHERONI and ENRICO BOTTONE, Riv. clin. pediat. **50**:145 (March) 1952.

After a rapid revision of the main points of lipid nephrosis, the literature concerning the use of corticotropin in the disease is taken into consideration. A case is reported in which corticotropin therapy was given and in which all the most important laboratory tests were made. The results obtained and the probable mechanism of the action of corticotropin are discussed.

FROM THE AUTHORS' SUMMARY.

PROTEIN AND SALT METABOLISM IN LIPOID NEPHROSIS. RUDOLF THURAU, Monatsschr. Kinderh. **97**:397 (Dec.) 1949.

Urinary sodium chloride varies inversely with proteins. During diuresis, salt excretion rises while protein excretions fall. The same relationship exists in the blood. When edema increases, protein excretions fall and NaCl excretion rises. When diuresis takes place, protein excretions rise and NaCl excretion falls. It is postulated that NaCl is used to maintain osmotic pressure in the blood and tissues to compensate for the hypoproteinemia. Urinary uric acid is always increased, while urinary urea and amino nitrogen are normal. Serum amino nitrogen is increased with edema and drops with diuresis. Nitrogen balance is negative during edema, and remains positive following diuresis. Anemia is not present despite low protein, as there is enough albumen present for hemoglobin synthesis.

HARTENSTEIN, Madison, Wis.

TREATMENT OF NEPHROSIS IN CHILDHOOD WITH MALARIA INOCULATIONS. FRIEDRICH LINNEWEH, Monatsschr. Kinderh. **99**:172 (May) 1951.

Repeated intravenous injections of Plasmodium vivax did not produce any favorable results in four children with nephrosis. This method of treatment was attempted in view of the favorable effects of various infections on the course of nephrosis.

HARTENSTEIN, United States Army.

ECTOPIA TESTIS. A. H. SMOOK, Nederl. tijdschr. verlosk. en gynaec. **9**:498 (March) 1952.

Among the deviations from the normal position of the testis, ectopia testis inguinalis interstitialis, that is, location of the testis on the aponeurosis of the musculus obliquus externus, is fairly frequent. In such cases hormonal treatment is useless. Early diagnosis is necessary. Among 25 cases of nondescended testicles in which operation was performed, ectopia was found in 12.

VAN CREVELD, Amsterdam, Netherlands.

Diseases of Ductless Glands; Endocrinology

THE ETIOLOGICAL SIGNIFICANCE OF ERGOT IN THE INCIDENCE OF POSTPARTUM NECROSIS OF THE ANTERIOR PITUITARY. GEORGE NASSAR, AIDA DJANIAN, and WILLIAM SHANKLIN, Am. J. Obst. & Gynec. **60**:140 (July) 1950.

It was observed that in cases in which ergotamine tartrate was administered for a few days because of postpartum hemorrhage symptoms suggesting ergotism developed. In the further course, symptoms indicating damage to the hypophysis became manifest. Experiments were undertaken to see whether a relationship exists between massive loss of blood and administration of ergot in the production of lesions of the hypophysis. It was found that rats weighing 50 gm. succumbed to an intraperitoneal injection of 7.2 mg. of ergotoxine ethanesulfonate in three hours. The solvent in the amounts used proved innocuous. (The solvent used was not indicated.) By cardiac puncture rats were bled of 1.4 to 3.0 per cent of their total body weight, which resulted in shock, and they were killed 2 to 10 days later. Only once did the anterior lobe of the hypophysis show signs of congestion. The hypophysis of all rats bled and given the ergot preparation intraperitoneally showed foci of necrosis limited to the anterior lobe. All rats not bled but receiving ergot in larger doses also had areas of necrosis in the anterior lobe, which was totally destroyed in one case. Changes in thyroid and adrenal glands were seen only in the latter group of rats and only rarely.

AMBERG, Rochester, Minn.

PRECOCIOUS SEXUAL DEVELOPMENT: A STUDY OF THIRTY CASES. GEORGE H. LOWREY and THELMA G. BROWN, J. Pediatrics **38**:325-339 (March) 1951.

The authors have reviewed the cases of 30 children with precocious sexual development. Females outnumbered males in a ratio of 9 to 1. The causes of precocious sexual development in this group are (1) the adrenogenital syndrome (nine cases, including one in a newborn infant with adenocarcinoma of the adrenal cortex); (2) central nervous system lesions (eight cases); (3) physiologic early puberty (seven cases), and (4) a miscellaneous group including polyostotic fibrous dysplasia (two cases), benign breast hypertrophy (two cases), Cushing's syndrome (one case), and one unclassified case.

MACDONALD, Pittsburgh.

PLAN OF RESEARCH ON ENDOCRINAL CONTROL OF HEMATOPOIESIS. G. CARDINI and M. VERROTTI, Clin. Pediat. **32**:596 (Nov.) 1950.

After reviewing the literature on hematopoiesis and the effect of the hormones of the endocrine glands on the disease, the authors announce that they will shortly publish four articles on the effect of (1) the sex hormones, (2) the pituitary hormones, (3) the adrenal cortex hormones, and (4) the thyroid, parathyroid, and thymic hormones on healthy children. The studies will be based on peripheral blood and bone-marrow studies before, immediately after, and one month after 10 days' medication with the individual hormones.

HIGGINS, Boston.

SPONTANEOUS HYPOGLYCAEMIA AND HYPERINSULINISM IN INFANTS AND CHILDREN. C. VAN BEEK, Maandschr. kindergeneesk. **20**:141 (June) 1952.

The author discusses the definition, symptomatology, classification, pathology, diagnosis, treatment, and prognosis of spontaneous hypoglycemia and hyperinsulinism in infants and children.

The physiological neonatal hypoglycemia is discussed, and an analysis is made of the recurrent type of severe hypoglycemia in children accompanied with attacks of convulsions or unconsciousness. This clinical entity is probably due to an abnormally slow transition from the newborn status to that of the normal child (Hartmann). The final prognosis is favorable.

The differential diagnosis between hyperinsulinism and other forms of spontaneous hypoglycemia is considered, and that between functional and organic hyperinsulinism. A classification is made of organic hyperinsulinism in hypertrophy and/or hyperplasia of the islands of Langerhans and in islet-cell tumors.

The newborn infant of the diabetic mother is discussed, in view of a large autopsy experience. During the first days of life a pathological hypoglycemia may exist. It is the clinical or merely the biochemical manifestation of a hypertrophy of the islands of Langerhans, which may often be so obvious that the islets are visible to the naked eye. The existence of a hyperplasia of islet tissue is, however, not yet proved. The hypertrophy of the islets is considered to be the morphologic answer to the stimulation caused by the fetal hyperglycemia. Thanks to a pathological-anatomical syndrome, which has been observed in 1939, the existence of maternal diabetes was diagnosed by autopsy on two stillborn children as well as on one newborn infant. This diagnosis was clinically confirmed.

In hydrops fetus universalis and icterus gravis neonatorum a mild or moderate hypertrophy of the islands is inconstantly present.

The author discusses the diagnosis of an insulin-producing islet-cell adenoma of the pancreas, a rare pathological condition in children, causing progressive paroxysmal hypoglycemia. For several reasons surgical exploration has to be performed as soon as possible. Adenomectomy results regularly in a dramatic cure.

Those cases of progressive paroxysmal hypoglycemia in which no tumor could be found on exploratory operation are considered. Frequently a subtotal pancreatectomy was performed, often with disappointing results. One has to give due consideration to the possibility that a small adenoma located in the head of the pancreas, or ectopic, is overlooked in these cases; it seems, however, that there is still greater possibility that they are cases of beta-cell hyperplasia; in this case treatment with corticotropin might be effective.

Special staining methods on alpha and beta cells are urgently needed in every case of organic hyperinsulinism.

FROM THE AUTHOR'S SUMMARY.

Diseases of Muscles, Bones, Joints, and Lymph Nodes

CARDIAC INVOLVEMENT IN PROGRESSIVE MUSCULAR DYSTROPHY. S. WEISENFELD and W. J. MESSINGER, Am. Heart J. **43**:170 (Feb.) 1952.

The report is based on a study of 44 patients with progressive muscular dystrophy. About 85% of the patients had some clinical manifestation of dysfunction in the cardiovascular system, and 80% had anomalies of the electrocardiogram.

Of 15 patients who died, postmortem studies were done on 6. The similarity of the lesions of cardiac and skeletal muscle was notable.

GIBSON, Chicago.

EWING'S ENDOTHELIAL MYELOMA OF ADOLESCENTS: REPORT OF TWO FATAL CASES. HORACE K. GIFFEN, Arch. Path. **46**:68 (July) 1948.

Ewing's tumor of bone, which he called "diffuse endothelioma of bone," occurs mainly in children. Two fatal cases in adolescents with typical clinical, histologic, and roentgenologic characteristics are presented. One patient was a 13-year-old boy with the original lesion in the upper femur; the other, a 16-year-old girl with the initial tumor in the scapula. Both were apparently in good health prior to onset of the disease, which was brought to attention after activity. Neither showed clear evidence of direct trauma. The author believes that this type of tumor is a cancer of the reticuloendothelial system which is located in the medullary regions of bones, especially in the diaphyses of long bones.

HAW CHAN, San Francisco.

DYSCHONDROPLASIA WITH HEMANGIOMATOSIS (MAFFUCCI'S SYNDROME) AND TERATOID TUMOR OF THE OVARY. J. F. KUZMA and J. M. KING, Arch. Path. **46**:74 (July) 1948.

The combination of dyschondroplasia and hemangiomatosis is rare. It was first described by Maffucci in 1881 and hence bears his name. Twenty-one cases have been reported in the literature. Dyschondroplasia (called Ollier's disease when occurring alone) is a nonhereditary mesodermal dysplasia affecting the growing ends of bones. The child is apparently normal at birth, but at puberty hard nodules on the fingers or toes, legs and arms, vertebrae, ribs, and scapulae appear. The skull, instep, and wrist are rarely involved. These nodules are identified as enchondromas. At the same time bluish tumors (hemangiomas) may develop on the affected limbs. Large dilated veins may be associated with the soft tumors. Distribution of the painless tumors may be asymmetric, but they are rarely unilateral. Skeletal development may be retarded, trivial injuries may cause fractures of long bones with delayed callus formation or nonunion, and deformities may be so grotesque that all function is lost and amputation becomes imperative for comfort. In the early twenties the condition becomes stationary. Chondrosarcoma has been noted in 4 cases in the literature. Other associated changes reported include abnormal sweating, vitiligo, and cerebral glioma. The case of a 19 year old girl is reported here, presenting the features of Maffucci's syndrome. In addition the patient had an ovarian tumor which was removed surgically and diagnosed variously by different pathologists as typical granulosa cell tumor, atypical arrhenoblastoma, mesonephroma, and teratoma. An x-ray diagnosis of pituitary tumor was also made. The patient died three years after operation with what was believed to be metastasis of the ovarian tumor. No autopsy was performed.

HAW CHAN, San Francisco.

CONGENITAL MALFORMATION OF VERTEBRAE (HEMIVERTEBRAE) WITH APLASIA OF CORRESPONDING RIBS, ASSOCIATED WITH A LATERAL MENINGOMYELOCELE: REPORT OF A CASE. I. RALPH GOLDMAN, Arch. Path. **47**:153 (Feb.) 1949.

Congenital absence of one or more ribs, accompanied or not with hemivertebrae, is very rare, especially in combination with a lateral meningomyelocele. Other congenital anomalies may or may not be present. The case reported is that of a full-term white boy who died the second day after delivery. There was no familial history of similar defects or prenatal infection. At autopsy there was found a lateral meningomyelocele with absence of the right halves of the first through ninth thoracic vertebrae and of their transverse processes. The remaining right ribs were present but were fused posteriorly. Other anomalies noted included a clubbed right foot, congenital dislocation of the hip, absence of the renal artery, renal vein, kidney, and ureter, on the right, megaloureter on the left, undescended right testis, and smaller right adrenal. The author utilizes the theory of Stockard to explain the thoracic irregularities. Stockard believed that in the development of every organ or part there is a critical stage characterized by rapid cell multiplication during which it is dominant over nearby organs. If during this critical period an environmental change acts adversely on the dominant organ, it may lose its dominance to other organs. Goldman believes that the adverse stimulus responsible for the formation of hemivertebrae may act on other developing organs during their critical period to produce concomitant defects. The defect of the neural arches in this case allowed the meninges to herniate into the posterior chest wall, forming the meningomyelocele.

HAW CHAN, San Francisco.

CLINICAL ASPECTS OF A CASE OF AMYOTONIA CONGENITA. E. ORLANDY, Lattante **22**:170 (March) 1951.

A case of amyotonia congenita is reported in a girl 8 months old. She was one of twins, her brother being perfectly normal. The amyotonia was present from birth. The differential diagnosis between amyotonia congenita and Werdnig-Hoffman syndrome is discussed.

HIGGINS, Boston.

THE PROGNOSIS IN INFANTS WITH CONGENITAL DEFORMITY OF THE HIP COMPLICATED WITH OSTEOMYELITIS. RAUL M. DEL CAMPO, Acta pediat. espãn. **9**:1423 (Dec.) 1951.

The author points out that hip subluxation existing in the presence of osteomyelitis is not a simple problem. Surgical intervention is necessary, and the chances of a final result without deformity are remote, even with prolonged immobilization.

After the cessation of the infection, special orthopedic measures may necessitate a long invalidism. Ankylosis may be the only course of action.

The usual Whitman treatment may fail because of the bone destruction, such as that of the femoral epiphysis and condyles.

ALLEN, San Francisco.

EFFECT OF CORTICOTROPIN (ACTH) IN A CASE OF JUVENILE RHEUMATOID ARTHRITIS. P. ASTRUP; K. BROCHNER-MORTENSEN; V. FABER; C. HAMBURGER; N. HARBOE; K. SCHMITH; E. SNORRASON; M. SPECHLER, and J. VESTERDAL, Acta paediat. **39**:215 (No. 3) 1950.

A 10-year-old girl with rheumatoid arthritis was treated with corticotropin. At first a marked clinical improvement was seen, as well as a decrease of the sedimentation rate to the normal. The treatment was continued with very small doses (2 mg. daily) for a month. The sedimentation rate soon rose to the initial value, but a clinical relapse was not seen before the end of this period. A new treatment with high doses (but with another batch of corticotropin) had only poor effect, and there was no decrease in the sedimentation rate.

After a few days' treatment albuminuria developed, and soon a severe nephrosis.

During the treatment the serum protein (particularly the α globulin), the serum uric acid and nonspecific hyaluronidase inhibitor in serum decreased. Hyperglycemia and glycosuria occurred to a slight extent. There were no significant alterations of serum K, Na, Ca, Cl, blood cholesterol, phosphatase, urea, plasmin, plasminogen, or antiplasmin.

The hemoglobin, the white blood count, and the number of circulating lymphocytes increased, while the eosinophiles decreased.

There were no significant alterations of the nitrogen balance, or in the excretion of creatin and creatinine. As to the excretion of K, Na, Ca and Cl, the results were inconclusive. The excretion of free hexosamine in the urine increased.

During the first period of high dosage the excretion of both 17-ketosteroids and glucocorticoids increased, but during the last period, when almost no clinical effect was seen, only the former increased.

FROM THE AUTHORS' SUMMARY.

Diseases of Eye

STRABISMUS IN SCHOOL CHILDREN. JOSÉ AGUILAR, Acta pediat. españ. **8**:373 (April) 1950.

Dr. Aguilar, in this issue devoted to the health of the school child, reports that he examined 288 children (170 boys and 118 girls) referred to him by the school division of the State Health Department. These fell into three groups: children with normal vision, 15; children with infections but with normal visual apparatus, 82, and children with refractive errors, 191.

Of the 191 with refractive defects, 153 had myopia, hypermetropia, or astigmatism, and 38 had strabismus. Strabismus was of three types: strabismus due to central nervous system lesion, 8 cases (21%); strabismus with myopia, 10 cases (26%) and strabismus with hypermetropia, 20 cases (53%).

The central nervous system lesions are the least amenable to treatment. Lesions occur in the nuclei of the third, fourth, or fifth cranial nerve and may be associated with mental deficiency. Other ocular defects are noted, including ptosis and nystagmus. The author discusses the relation of this type of strabismus to the Hertwig-Magendie type and to the Tay-Sachs syndrome. He also links the lipid infiltration in such diseases as Gaucher's, Neimann-Pick and Hand-Schüller-Christian disease with this rather hopeless type of strabismus.

The treatment in the other types is much easier, although the myopia with strabismus is more difficult than with hypermetropia. The author concludes that strabismus in hypermetropia is most common; that strabismus is usually divergent in myopic children and convergent in hypermetropia; that strabismus should be corrected as early as possible with spectacles, and that in a large number of cases the strabismus may be permanently corrected with early use of the proper correction.

ALLEN, Santa Fe, N. M.

NEW OBSERVATIONS ON THE ORIGIN OF SQUINT. G. B. J. KEINER, Maandschr. kindergeneesk. **19**:217 (Sept.) 1951.

Many theories have been advanced as to the origin of squint, but not one of them has been able to withstand thoughtful criticism. Zeeman's ideas on the development of the optomotor reflexes, which opened up new perspectives, formed the basis for a study of the development of these reflexes in normal and squinting children and in children with "myelogenesis retardata." In this connection it was first necessary to ascertain the age at which the deviation first appears.

Contrary to the accepted opinion, it was found in a study of 656 very young children that this occurred before the end of the first year in 54% and before the end of the second year in 78% of cases. The frequency curve showed that all infants are apparently born with a tendency to squint, but that corrective factors leading to orthophorization make their appearance at the age of about 6 months.

An investigation of the optomotor reflexes in young squinting children showed that here, in contradistinction to the normal development, stimulation of the temporal half of the retina generally produced monocular adduction, while stimulation of only the nasal half in some cases gave rise to a conjugate movement.

There was thus a disturbance of the harmonious development of the optomotor reflexes. The cause of this is not in the retina, as was shown by the electroretinogram taken in a number of cases. It is suggested that the cause of the disturbance which leads to a predominance of monocular adduction, and hence to a squinting position, may be a retarded maturation and myelination of certain optomotor paths. Support for this view was obtained from the examination of a number of children with "myelogenesis retardata," in all of whom strabismus developed later.

The possibility of a hormonal cause of squint is suggested.

FROM THE AUTHOR'S SUMMARY.

Cutaneous Diseases; Allergy

TREATMENT OF RESPIRATORY ALLERGY IN CHILDREN BY ORAL ADMINISTRATION OF DUST AND POLLEN EXTRACTS. J. HARVEY BLACK and JAMES HOLMAN, J. Allergy **21**:148 (March) 1950.

Oral treatment for allergy was given to 402 children under 13 years of age. Adequate follow-up records were not available on 108 of these children; they are not included in the report. Pollen treatment was done with a 2% glycerosaline solution similar to that used for hypodermic treatment. Dust treatment was done with a concentrated glycerosaline solution similar to the one used for hypodermic treatment.

One drop of the solution was given in six to eight ounces of water, milk, or orange juice between meals. The dose was increased 1 drop each day until 15 drops were given. If relief was obtained, this dose would be continued. If there was no evident relief by the time 15 drops were given, this dose would be continued for a week; if no relief was obtained at the end of the week, oral therapy would be discontinued and hypodermic treatment suggested.

Oral therapy was successful in more than one-half of the cases. Hypodermic treatment gave relief to a large number of patients who failed to get relief with the oral method.

HOYER, Cincinnati.

ROENTGEN STUDIES OF ALLERGIC CHILDREN WITH DISTURBANCES OF THE PYLORUS RESULTING FROM FOOD SENSITIVITY. JOSEPH H. FRIES, J. Allergy **23**:39 (Jan.) 1952.

Fluoroscopic examinations and roentgenograms were made after the ingestion of barium solution containing a food to which the patient was sensitive. The reaction occurred most often in the pyloric area of the stomach, evidenced by a blockage in the pylorus with retention of the barium solution in the stomach. The blockage of the pylorus disappears with the termination of the allergic reaction. The abnormalities revealed by the roentgenogram in the pylorus resemble those of organic lesions. However, they were shown to be transient. The possibility of allergic involvement must be considered in the interpretation of abnormalities of the pylorus, as revealed on the roentgenogram.

HOYER, Cincinnati.

ERYTHEMA MULTIFORME EXUDATIVUM. LYDIA WRIGHT and SAMUEL JENKINS, J. Pediatrics **38**:310-314 (March) 1951.

Wright and Jenkins report two cases of erythema multiforme exudativum. One, representing the severe pluriorificialis type, was quickly cured by aureomycin when penicillin had apparently failed. The other, a mild but recurrent case, with only a skin eruption, did not respond to penicillin, aureomycin, or local applications of sulfathiazole. The authors feel that the two variants of the disease are separate entities and plead for a sound classification of this disease and its variants.

MACDONALD, Pittsburgh.

VITAMIN B_{12} IN DESQUAMATIVE ERYTHRODERMA—LEINER'S DISEASE. L. SALMI and L. MARTONI, Clin. pediat. **32**:628 (Nov.) 1950.

Four infants with Leiner's disease, each about 2 months of age, showed a very satisfactory therapeutic response to vitamin B_{12} administration (7 γ parenterally on alternate days).

HIGGINS, Boston.

STURGE-WEBER DISEASE. G. CARDINI, Lattante **22**:158 (March) 1951.

A male infant with a large congenital birthmark over the right side of the face and scalp was apparently quite well until he was 5 months old, when he had two severe left-sided convulsions lasting 6 and 40 minutes. Lumbar puncture was negative. A diagnosis of Sturge-Weber disease (nevoid amentia) was made. The basis of the symptoms in this condition is discussed.

HIGGINS, Boston.

THERAPEUTIC VALUE OF SOME OF THE B VITAMINS AND OF EMBRYONIC LYSATES IN INFANTS WITH DESQUAMATIVE ERYTHRODERMIA. T. PORCELLI, Pediatria **60**:56 (Jan.) 1952.

On the basis of a study of 64 infants with desquamative dermatitis, vitamin B_6 proved the most efficacious therapy. Some of the children seemed to benefit when amino acids were given.

HIGGINS, Boston.

Surgery and Orthopedics

BRACHIAL PLEXUS BLOCK ANESTHESIA IN CHILDREN. GEORGE A. SMALL, J. A. M. A. **147**:1648 (Dec. 22) 1951.

In 150 children ranging from 15 months to 12 years brachial plexus block anesthesia was used for various surgical procedures. In 125 the results were good and rapport was established. The procedure required 40 minutes, on the average, to execute, including premedication with hypnotics. The details of technique are described.

KIMBALL, Evanston, Ill.

STATISTICAL CONSIDERATIONS ON FLAT FEET. CARLOS A. BAUZA and GALINA SOLOVEY DE MILECHNIN, Arch. pediat. Uruguay **22**:5 (Jan.) 1951.

The problem of the physical and mechanical changes in the feet interests not only the specialist in orthopedics but also the general pediatrician. The latter must appreciate their incidence and carry out the work of hygiene and prevention, while the former enters more deeply into the study of the feet.

The diagnosis must be clinical and roentgenological and must be made with footprints, since any one of these procedures is by itself incomplete. The footprint method is useful for statistical study. Although they have made only footprints, Bauza and de Milechnin point out that other authors have obtained very valuable information from prints of the calf and heel in measuring the pronation of the foot. Of the different measurements that can be taken of the footprint Bauza and de Milechnin have chosen Schwartz's footprint angle.

They find that the largest number of cases show values around 50 degrees, and that the average measurement of the angle is smaller (indicating flatter feet) in little children, increasing gradually with age, and that the percentage of angles smaller than 43 and 33 degrees is 25.2 and 11.5 in boys, and 25.1 and 6.2 in girls. A slight lack of symmetry is usual.

FROM THE AUTHORS' SUMMARY.

Teeth and Dentistry

THE EFFECT OF TOPICALLY APPLIED FLUORIDES ON DENTAL CARIES EXPERIENCE. JOHN W. KNUTSON and GRACE C. SCHOLZ, Pub. Health Rep. **64**:1403 (Nov. 11) 1949.

New caries in children's teeth was decreased by topical application of 10 to 12% sodium fluoride in the first molars and 16 to 23% in second molars. In the other teeth the percentage of caries was very small and the difference between the treated and untreated groups was too small to evaluate.

KIMBALL, Evanston, Ill.

Miscellaneous

POISONINGS COMMON IN CHILDREN. J. R. ROSS and ADAM BROWN, Canad. M. A. J. **64**:285 (April) 1951.

Cases of poisoning in children admitted to the Hospital for Sick Children, Toronto, over a 10-year-period are summarized. Fourteen per cent of the patients died. Lead was the commonest poison, with strychnine next. Other toxic agents were illuminating gas (10 cases with 2 deaths); morphine (4 cases with 2 deaths); methyl salicylate (oil of wintergreen) (2 cases with 2 deaths); poison oak (1 case with 1 death); chloral hydrate (1 case with 1 death); lemon oil furniture polish (1 case with 1 death); aspiration pneumonia due to inhalation of zinc stearate (4 cases with 2 deaths); aspiration pneumonia (7 cases with 1 death); and aspiration pneumonia due to Sloan's liniment® (1 case with death). Nonfatal poisonings included 10 due to atropine, 8 to mercury, 5 to food poisoning, 5 to phenobarbital (luminal®), 5 to lye, 4 to acetanilid, 4 to arsenic, and 3 to aniline dye. Lead poisoning was studied separately and was the commonest and caused the most deaths with 23 cases in two years and 5 deaths.

MCGUIGAN, Evanston, Ill.

NOTES ON THE HISTORY OF PEDIATRICS IN SCOTLAND. H. P. TAIT, Edinburgh M. J. **58**:180 (April) 1951.

Tait has traced the development of child health services in Scotland through the years. Active interest in child health really started at about the end of the 18th century. Orphan homes, industrial schools, and homes for handicapped children were developed. The first chair of midwifery was established at Edinburgh in 1726. Obstetricians gradually began to replace the midwives and the "wise-women"; the latter were persons who warded off evil spirits by charms and incantations and were the constant advisors of the mothers.

Interesting highlights in this history include a 13th century ordinance against the practice of mothers' taking their infant children to bed with them; this law was aimed at prevention of smothering or "smooring" of infants, a recognized practice that, despite the ordinance, continued into the 19th century, especially among poor and unmarried mothers.

One of the first stations for Jennerian vaccination in Britain was opened in Glasgow in 1801. At least a century before that the Highland Scots had practiced a modified form of vaccination, infecting their children "by rubbing them with a kindly pock."

Scarlet fever was first recognized in Scotland in 1684; measles in Edinburgh in 1735; diphtheria (first called malignant angina) in 1758. The high death rates associated with these diseases led to various attempts at control, and in the latter part of the 18th century Francis Home reported a somewhat successful method for prevention of measles, applying the principles of smallpox vaccination. Another formidable disease, whooping cough (kink-cough), received attention from Robert Watt in 1813.

James Monteith gave courses of instruction in child health in Glasgow in 1778, and in the latter years of the 18th century Alexander Hamilton gave some instructions on children's diseases in the midwifery course at Edinburgh. In 1837 William Campbell set up extra-academic courses in children's diseases at Queen's College. Such side-courses continued, and recognition of the importance of pediatrics increased. The first children's hospital was organized at Edinburgh in 1860, and in 1885 the university formally granted the status of lecturer to two physicians on the staff of the hospital.

In Edinburgh, Glasgow, Aberdeen, and Dundee pediatric education has advanced through the years, aided by the efforts of many illustrious Scots. Perhaps the greatest of these was John Thomson of Edinburgh, who died in 1926.

MACDONALD, Pittsburgh.

CASE REPORTS. Bol. méd. Hosp. inf. **7**:390 (June) 1950.

CASE 1.—A case of fibrocystic disease in a 6-month-old girl is presented; the picture corresponds to Dr. S. Farber's group 2. Death was caused by an acute respiratory process.

CASE 2.—A case of malignant lymphoma in a 4-year-old boy is presented. The process affected the mesenteric lymphatic glands, and totally invaded the pancreas; a partial invasion was found in other organs such as duodenum, stomach, ileum, transverse colon, kidneys, perisuprarenal fibroadipose tissue of the right side, diaphragm, thyroid, and hypophysis.

CASE 3.—A case of intestinal infarction in a 14-month-old girl is reported. The infarct may be explained by a probable localized venous thrombosis in situ, possibly caused by enteritis.

CASE 4.—A case of tuberculous meningoencephalitis in an 8-year-old girl is presented. The process caused internal hydrocephalus by obstruction of the Sylvian aqueduct; also there were focal lesions on the left cerebral cortex and on the right cerebellar hemisphere.

CASE 5.—Presentation is made of a case of astrocytoma of the left cerebellar hemisphere with a great cystic formation which compressed the fourth ventricle, causing a slight internal hydrocephalus by obstruction of the ventricular system; it also injured the pyramidal system, probably by compression; also, the right tonsilla cerebelli suddenly compressed the bulb and caused a final respiratory failure.

CASE 6.—The last case deals with a hemolytic disease caused by a slight degree of the Rh factor; it was complicated by omphalitis, which was cured. Primary atypical pneumonia and a slight rachitism were also present.

FROM THE AUTHORS' SUMMARY.

CASE REPORTS. M. S. MARTINEZ, A. ESCOBAR, A. R. GAMBOA, G. POUMIAN, P. DE M. LEONARDO ARZOLARZA, and JULIO C. RASCON, Bol. méd. Hosp. inf. **7**:553 (Aug.) 1950.

CASE 1.—Presentation is made of a case of atresia of the esophagus in a boy 5 days of age. The immediate cause of death was aspiration pneumonia with a superadded bronchopneumonia.

CASE 2.—The author reports a case of septicemia by a species of Neisseria in a 9-year-old girl. Death was probably due to pneumonia added to a cardiopathy.

CASE 3.—The author presents a case of diffuse hepatic necrosis accompanied by a hemorrhagic state in a 4-month-old boy. The author considers that death might be imputed primarily to a probable acute hepatic insufficiency to which a terminal factor might be added.

CASE 4.—A case of cyanotic congenital cardiopathy in a 2-month-old girl is presented. The immediate cause of death seemed to be an obliteration of the arterial duct, since in this case it was the only connection with the lesser circulation.

CASE 5.—Report is made of a case of fibrocystic disease in a girl 5 months old. There also was nephrosis of unknown etiology. A respiratory infection was probably the ultimate cause of death.

CASE 6.—The author discusses a case of medulloblastoma in a 5-year-old boy. The disease brought about an obstructive condition of the ventricular system which caused an internal hydrocephalus. The patient died from suppurative meningitis and embedding of the tonsillae cerebelli.

FROM THE AUTHORS' SUMMARY.

A CASE OF ANGIOEPITHELIAL SARCOMA IN AN INFANT OF 40 DAYS WITH FAVORABLE COURSE. C. CHOREMIS and B. KYRIAKIDES, Helvet. paediat. acta **7**:202 (April) 1952.

A case of multiple angioendotheliosarcomas of the skin and bones, accompanied with pathologic fractures, is reported in a 6-week-old infant. After biopsy of a tumor of the skin over the ankle, all tumors spontaneously regressed without any treatment; they had completely vanished at the end of another four months.

FROM THE AUTHORS' SUMMARY.

SIXTH INTERNATIONAL CONGRESS OF PEDIATRICS. Schweiz. med. Wchnschr. **80**:29 (July 22) 1950.

This number is devoted to the Sixth International Congress of Pediatrics. The first report, pp. 744-745, by J. P. Hubbard, deals with the improvement of pediatric care in relation to public health. A survey conducted by the American Academy of Pediatrics showed the extent to which pediatric practice in the United States has shifted from the care of the sick to health supervision of well children. Pediatric training in medical schools and in the hospital services for interns and residents has not kept in step with this change.

The care of the premature infant as a problem of world health is discussed by F. C. Dunham, pp. 745-751, who reports the recommendations of a group of experts whose conference was sponsored by the World Health Organization in Geneva this spring.

Active immunization against infectious diseases is the subject treated by Paul Artom di Sant' Agnese, pages 749-751. Owing to the conditions prevailing in the Northeastern United States, and particularly in New York, the immunization of infants and children against pertussis, diphtheria, and tetanus is given special attention. If pertussis vaccine alone is given it is recommended that the first injection be given during the second or third month of life. If it is given together with other antigens it is recommended that the injections be started when the infant is 3 months old. But it has been demonstrated that the great majority of infants who received the first injection at the first week, the second at the fifth and the third at the ninth week produced antibodies against pertussis, diphtheria, and tetanus in amounts sufficient to protect against these infections. The maintenance of the immunity by means of repeated injections at given periods is discussed.

C. E. Dent gives a very interesting presentation of amino acid metabolism (p. 752). An attempt has been made to summarize the theoretically possible mechanism by which abnormal accumulations of amino acids, or of their immediate degradation products, could occur in body fluids and tissues. Three basic mechanisms are distinguished: a "metabolic block," where one or more enzymes are absent; a "deviation of metabolism," where the enzymic machinery is present but is not used adequately, and an "excretion defect." As example of the "metabolic block" phenylpyruvic oligophrenia may be mentioned. Here phenylalanine is accumulated in the body, since the oxidation of the benzene ring of the molecule is interfered with. The alanine part of the molecule is changed by the kidney to pyruvic acid, so that phenylpyruvic acid is excreted and traces of phenyllactic acid. Other disorders of the metabolism, such as inhibition of melanin formation from dihydroxyphenylalanine (dopa), alkaptonuria, and renal aminoaciduria with cystinuria, are also discussed.

The role of potassium in the clinic is rather briefly presented by C. Darrow (p. 756). In cases of acidosis accompanying infantile diarrhea a solution containing 120 mEq. sodium, 35 mEq. potassium, 102 mEq. chloride, 52 mEq. bicarbonate or lactate per liter is parenterally administered. The solution contains sufficient potassium for a day in 80 ml.

C. Fanconi presented a paper entitled "New Aspects of Renal Pathology: (1) Chronic Aminoaciduria; (2) Diabetes Salinus Renalis; (3) Lower Nephron Nephrosis" (p. 757). Some

diseases, such as renal glycosuria and particularly chronic aminoaciduria, are attributed to an insufficiency of the proximal parts of tubules. An insufficiency of Henle's loop leads to salt deficiency due to diabetes salinus renalis. An insufficiency of the distal tubules produces lower nephron nephrosis.

In "Renal Function During the First Days of Life" (p. 762) R. A. McCance gives a rather short review, referring for greater detail to other publications.

M. Viscontini describes antivitaminic activity and its mode of action (p. 763) from the chemical point of view. An antivitamin is characterized as a substance which can block the action of a vitamin specifically and reversibly.

The pharmacology of vitamin D with special consideration of D-hypervitaminosis is discussed by G. O. Harnapp (p. 765). The author speaks in the main of D_2 (calciferol). The daily antirachitic requirement is given as 80 to 400 I.U. The international unit is equivalent to 0.025 γ of the crystallized substance. For mass prophylaxis a dose of 15 mg. is proposed. The dosage usually employed in the treatment of rachitis is harmless. With "high dosage" lesions are unavoidable. The lesions are described. So far 11 fatal results have been reported. All symptoms of D hypervitaminosis in man disappear nearly without exception.

G. Miescher gives a general view of eczema (p. 771). The primary and most important lesion is the vesicle derived from circumscribed intercellular edema (spongiosis). Not all dermatoses characterized by spongiosis are allergic manifestations.

Hans Debrunner, in the article entitled "Congenital Clubfoot" (p. 772), concerns himself particularly with genetic factors.

The number concludes with "Acute Psychoses Accompanying Somatic Diseases and Schizophrenia in Childhood" (p. 774), by Jakob Lutz. Psychoses are seen in children associated with various infectious-toxic diseases of the body, particularly with lesions of the brain and its coverings. The psychosis is a symptom of the disease and disappears with it. It is called exogenous or symptomatic. These psychoses belong to the acute exogenous reaction type (Bonhoeffer). The most important differential diagnosis is the distinction from childhood schizophrenia. This distinction is relatively easy in a few typical cases, but usually it is rather difficult. Schizophrenia in childhood is much rarer than the acute exogenous reaction, so it is stated that only 1% of all cases of schizophrenia fall in the first decade. If it is assumed that about 1% of the total population is schizophrenic, one child with schizophrenia will be seen among 10,000 persons. The acute exogenous reactions are much more numerous.

It is concluded that if acute psychosis in childhood is similar to adult schizophrenia, it is usually not schizophrenia but an exogenous reaction referable to a somatic disease. Schizophrenia in children is a rare disease. It presents a symptom picture which comprises the fundamental disorders of schizophrenia but is partly less unfolded and partly is expressed in a childish manner. And thus it is differentiated from the psychosis of the adult.

Lawson Wilkins writes on "Adrenocortical Diseases in Children" (p. 766). The relationship of the chemical structure to the effect of various products of the adrenal cortex is outlined. It is undecided as yet whether the different substances arise from one precursor or are synthetized independently. The adrenal cortical insufficiencies are classified and the treatment is indicated briefly. Hyperadrenocorticism may be divided into the androgenital syndrome and Cushing's syndrome. In the former there are excessive protein anabolism, growth and viralization; in the latter, protein anabolism and growth are inhibited, while gluconeogenesis is increased with a tendency to obesity. There is also some increase in androgen in Cushing's syndrome, but the predominating effects are due to excess of gluconeogenetic corticoids. Congenital adrenal hyperplasia causes pseudo-hermaphroditism in females and macrogenitosomia precox in males. Tumor or hyperplasia of the adrenal beginning postnatally causes virilization in both sexes. In males macrogenitosomia precox resembling that of the congenital type usually occurs. So far only one feminizing tumor has been described in childhood. The small uncapsulated tumor removed from this 5-year-old boy could not be differentiated from a virilizing tumor histologically. The treatment of adrenal tumors is surgical. The author discovered that the excessive secretion of androgen can be suppressed by small doses of cortisone. The paper should be consulted for much interesting detail.

AMBERG, Rochester, Minn.

CONCENTRATIONS OF PENICILLIN IN THE BLOOD SERUM OF INFANTS TREATED WITH VARIOUS-SIZED DOSES. M. MIDULLA, Arch. ital. pediat. e puericolt. **13**:203, 1949.

Midulla has experimentally studied the concentration in the blood serum of penicillin given intramuscularly in various-sized doses to children of different ages and weights; some of them were feverish and some had kidneys, liver or heart impaired. The author deems it possible to reach the proper concentration against common infections by administering 1,000 units of penicillin per kilogram of body weight every 3 hours; that is, 8,000 units per kilogram during 24 hours.

FROM THE AUTHOR'S SUMMARY.

MALIGNANT TUMORS IN CHILDHOOD. AAGE VIDEBAEK, Acta pædiat. **39**:502 (No. 6) 1950.

The incidence of malignant disease in childhood is about one to two cases per 10,000 children. About 4% of all deaths among children over one year of age are due to malignant tumors.

The lowest incidence of cancer for both sexes is in prepuberty, at about the seventh year of age. Actual childhood tumors occur before this age and are predominantly sarcoma and mixed tumors, which are interpreted as a separate group of a special pathogenesis thought to be independent of the hormonal level.

The few carcinomas which occur in childhood are chiefly epitheliomas; carcinoma of the alimentary tract is even more uncommon.

FROM THE AUTHOR'S SUMMARY.

UNDERGRADUATE TEACHING OF PEDIATRICS. ARVID WALLGREN, Acta pædiat. **39**:524 (No. 6) 1950.

The increasing importance of child health makes a revision of pediatrics in the medical curriculum necessary. Pediatrics should be made one of the main clinical subjects, and the teaching of pediatrics should be effective enough to guarantee satisfactory practical knowledge of the healthy and sick child. The importance of selection of the teachers and the students, restriction of the number of accepted students, organization of the teaching program, and better teaching technique are stressed. The individual bedside training in the wards or in the outpatient department should be more emphasized. The collective teaching in the classroom should be only a minor part of the program; it should be improved by the introduction of new methods aiming at a more active participation of the audience. The length of the pediatric curriculum should preferably be not less than three months, the optimal time probably being five months, including clerkship or internship. The course should be postponed until the end of the clinical undergraduate studies and terminated by an examination.

FROM THE AUTHOR'S SUMMARY.

POISONING OF A CHILD WITH SHOE POLISH. GEJZA CINOVSKY, Pediat. listy **4**:260 1949.

A 1½-year-old boy drank 1 dl. of a cobbler's shoe polish. He became unconscious, the skin and sclera were black, and he was in a state of collapse. The blood taken by venepuncture was blackish and had the smell of the shoe polish. Therapy included administration of glucose and cardiac stimulants. He was cured. The polish contained 40% nitrobenzol.

FROM THE AUTHOR'S SUMMARY.

THE ACTION OF COLA DRINKS WHICH CONTAIN CAFFEIN. D. A. A. MOSSEL, Nederl. tijdschr. verlosk. en gynaec. **95**:1357 (May 5) 1951.

To some cola drinks, prepared in the Netherlands, caffein is added in concentrations varying from 20 to 75 mg. per bottle of about 200 ml., while other such beverages lack caffein altogether. This highly undesirable situation must be cleared by adequate labeling of these types of beverages.

Since caffein is neither an essential constituent of a beverage nor a nutrient, the toxicology of its addition must be considered in the long range feeding experiments tentatively proposed by the United States Food and Drug Administration.

Such experiments have not yet been carried out, but it has been shown that an unfavorable gastric response may be expected.

As long as no proof is at hand that caffein stands the Food and Drug Administration test, children and patients with diseases of the stomach (as well as all those who are not allowed to take caffein) should be dissuaded from taking cola beverages containing caffein.

A further potential risk involved in the consumption of some cola beverages is dental erosion due to the presence of phosphoric acid and the very low pH, of about 2.5.

VAN CREVELD, Amsterdam, Netherlands.

A SIMPLE METHOD TO MAKE CASTINGS, ESPECIALLY OF HEART AND LUNGS. P. D. KAPSENBERG, Nederl. tijdschr. verlosk. en gynaec. **96**:493 (March 1) 1952.

A simple method is described to make models of the heart, the bronchial tree, and the pulmonary vessels; these are especially for use by thoracic surgeons.

VAN CREVELD, Amsterdam, Netherlands.

A STUDY ON THE ANTIBACTERIAL AND TOXIC ACTION OF A NEW MOLECULAR COMPOUND, PICRYL SULFACETAMIDE. T. K. CH'UAN and Y. P. WU, Chinese M. J. **67**:196 (April) 1949.

Picryl sulfacetamide is synthesized by adding trinitrophenol (picric acid) to sulfacetamide. In vitro, the antibacterial effect of this compound is high, but the drug is too toxic to be used as a therapeutic agent.

MACDONALD, Pittsburgh.

A TOXICOLOGICAL STUDY OF ANTIARIS TOXICARIA. T. K. CH'UAN and C. W. HAN, Chinese M. J. **67**:261 (May) 1949.

Nu-Chien-Tzu is a dried exudate of Antiaris toxicaria Lesch, which grows in drained sandy soil on Hainan Island. The name represents the constituents of an arrow poison used by the Miao race of Hainan inhabitants. It has been reported that this poison contains powerful cardiotonic glucosides. The authors' animal experiments confirm this idea. The drug is very toxic but is excreted and detoxified rapidly. It acts directly on the heart muscles and its action is more effective but more toxic than that of digitalis. Since it is eliminated rapidly, there is no cumulative effect. The cardiotonic action raises blood pressure, promotes the heart beat, and increases heat output. It is hoped that a further study of this drug may result in some finding of clinical significance.

MACDONALD, Pittsburgh.

ANTIBODIES AND PROTEIN NUTRITION. CHIEN-LUNG YI, Chinese M. J. **67**:340 (June) 1949.

The author asks the question, Does protein depletion or protein repletion change the capacity for antibody or gamma-globulin production in the body? He reviews the literature and answers that it does. He suggests that the high incidence of infectious diseases in periods of famine and starvation and the frequent intercurrent infections in chronic debilitated patients might be due to impaired antibody production as a result of low protein intake.

MACDONALD, Pittsburgh.

News and Comment

GENERAL

Quarterly Journal Devoted to the Newborn.—Attention is called to the recently launched *Neo-Natal Studies* or *Études Néo-natales* published in French and English under the sponsorship of the International Children's Centre in Paris. The editorial board includes three American investigators. The editorial policy is aimed at publication of review articles from pediatricians, obstetricians, pathologists, physiologists, and authorities in other fields selected for their bearing upon the fundamental problems of human adjustment from intrauterine to extrauterine life. The first number appeared in February, 1952. Subscriptions may be arranged through Lange, Maxwell & Springer, Ltd., 41 Neal St., London, W.C. 2, at £1 18*s.* 6*d.* (roughly $5.40) per year.

GENERAL NEWS

Specialists Certified by the American Board of Pediatrics, Inc.—The following physicians were certified as specialists by the American Board of Pediatrics, Inc., after examinations held in Boston on Dec. 5, 6, and 7, 1952:

Ahrens, Edward H., Jr., New York.
Albis, Francis Joseph, New Haven, Conn.
Anderson, Robert Harper, Alexandria, Va.
Annunziato, David, Massapequa, N. Y.
Appleton, Ruth, Boston.
Askue, William E., Elkins Park, Pa.
Berry, Abram, Charleston, S. C.
Boggs, Thomas Renwick, Jr., Philadelphia.
Booth, O. Watts, Newport News, Va.
Burge, Russell S., Hicksville, N. Y.
Bush, James A., Salt Lake City.
Caldroney, Thomas W., Fort Eustis, Va.
Cogan, Gilbert M., Lowell, Mass.
Cook, Harold R., Brooklyn.
Crouch, Walter Lee, Wilmington, N. C.
Downing, Daniel F., Philadelphia.
Dumars, Kenneth W., Jr., Colorado Springs, Colo.
Emanuel, Vera, Babylon, L. I., N. Y.
Engelman, Frank Ephraim, Pittsburgh.
Erhard, Loraine Holman, Clearfield, Pa.
Ferris, Emil Michael, Chicopee, Mass.
Filer, H. Lambert, Jr., New Haven, Conn.
Fomon, Samuel Joseph, Fort Dix, N. J.
Frankel, Jack, Forest Hills, L. I., N. Y.
Frick, Oscar Lionel, Huntington, L. I., N. Y.
Goldstein, Norman, Washington, D. C.
Guard, Harold L., Major (MC) U. S. A.
Haiken, Irwin B., Brooklyn.
Hainsworth, Winston Clarkson, Willimantic, Conn.
Hasegawa, Masato Matthew, Honolulu, T. H.
Heald, Felix P., Jr., Boston.
Hoffman, Charles, Levittown, L. I., N. Y.
Ianotti, William A., Astoria, L. I., N. Y.
Jaworski, Rudolf Alexander, Pawtucket, R. I.
Kaufman, Paul, Arlington, Va.
King, James Patrick, Boston.
McCurdy, Robert G., Atlantic Highlands, N. J.
Mache, Charles L., Charleston, S. C.
Michel, Lawrence I., New Haven, Conn.
Millner, Bernard N., Trenton, N. J.
Minor, James V., Jr., Norwalk, Conn.
Mitchell, Joe Elias, Capt. (MC), U. S. A.
Nau, Otto S., Jr., Arlington, Mass.
Orbeton, Everett Arnold, N. H.
Rosenthal, Irving A., Brooklyn.
Rowley, Samuel Dunham, Hartford, Conn.
Sappenfield, Robert Warren, Philadelphia.
Scannell, Robert John, Lowell, Mass.
Silverman, Mendel, Pittsburgh.
Suskind, Lieselotte, Buffalo.
Teiger, Paul, Waterbury, Conn.
Tepper, Clifford M., Schenectady, N. Y.
Weaver, James M., Fort Lauderdale, Fla.
West, Robert, Chappaqua, N. Y.
Williams, Robert D. B., Rochester, N. Y.

ANNOUNCEMENTS

Coming Examinations to Be Held by the American Board of Pediatrics, Inc.—The American Board of Pediatrics, Inc., will hold oral examinations on Feb. 20, 21, and 22, 1953, in Baltimore and March 27, 28, and 29, 1953, in Memphis.

Special Three-Day Postgraduate Courses in Clinical Rheumatic Fever and Rheumatic Heart Disease.—The Committee on Rheumatic Fever and Cardiac Disease of the American Academy of Pediatrics has made an effort to stimulate the organization of short courses in rheumatic fever and rheumatic heart disease for pediatricians, with major emphasis on the clinical aspects of the disease, a minimum of didactic lectures, and a maximum opportunity for case studies, auscultation, fluoroscopy, interpretation of electrocardiograms, etc. In response to this effort, two special postgraduate courses will be offered in the spring of 1953 as follows:

New York.—New York University Postgraduate Medical School: April 15, 16, and 17, daily 9 a. m. to 4 p. m. Minimum enrollment 8, maximum 16. Fee $35. For further information or enrollment address C. E. dela Chapelle, M.D., Associate Dean, New York University Postgraduate Medical School, 477 First Ave., New York 16.

Philadelphia.—Children's Hospital: April 24, 25, and 26, daily 9 a. m. to 5 p. m. Enrollment limited to 10. Fee $35. For further information or enrollment address Rachel Ash, M.D., Associate Professor of Cardiology in Pediatrics, Children's Hospital of Philadelphia, 1740 Bainbridge St., Philadelphia 46, Pa.

PERSONAL

Appointment for Dr. Leo J. Geppert.—Dr. Leo J. Geppert, chief of the pediatric service at Brooke Army Hospital, Fort Sam Houston, Texas, for the past seven years, has been transferred to the Tokyo Army Hospital for a three-year tour of duty as chief of pediatrics and theatre consultant in pediatrics.

CORRECTION

In the article by Albert Edward Sobel, Ph.D., and Abraham A. Rosenberg published in the November, 1952, issue, page 614, Table 3 should read as follows:

TABLE 3.—*Disintegration Studies of Multivitamin Capsules*

Capsule	Treatment pH	Treatment Temperature	Disintegration Time in Minutes * 1	2	3	Comment
A	2.0	37 C.	>310	..	..	Swelled to approximately 3 × size; did not rupture †
B	2.0	37 C.	45	70	80	Gray precipitate; turbid red supernatant; oil on surface †
C	2.0	37 C.	2	15	30	Yellow precipitate; turbid red supernatant †
A	7.8	37 C.	>210	..	..	See data for pH 2 (above)
B	7.8	37 C.	8	..	..	See data for pH 2 (above)
C	7.8	37 C.	3	..	..	See data for pH 2 (above)
A	7.8	37 C.	2.5	30	49	Gray precipitate; clear brown supernatant
B	20 mg. per 100 cc. trypsin and 23 mg. per 100 cc. chymotrypsin ‡		2.5	21	40	Surface oil phase; orange precipitate; turbid yellow supernatant
C			3.0	56	152	Yellow precipitate; clear red supernatant
A	2.4	.....	15	50	70	.....
B	20 mg./ml. of "1: 10,000" pepsin (Parke, Davis & Co.)		8	18	25	.....
C			8	>235	..	.....

* Column 1 indicates (1) beginning of disintegration of capsule covering; (2) spilling of contents into incubation medium, and (3) complete disintegration.
† Same treatment with mild agitation (Warburg) gave same results.
‡ Chymotrypsin and trypsin in physiological levels of duodenal juice according to Dr. H. Goldenberg.

The legend in the upper right corner of Chart 1 should be as follows: solid line, Capsule A; line of one long and one short dash, Capsule B; line of one long and two short dashes, Capsule C; line of short dashes, Ref. Oil.

Books

Textbook of Orthopedics. By M. Beckett Howorth, M.D. Price, $16. Pp. 1110, with 463 illustrations. W. B. Saunders Company, 218 W. Washington Sq., Philadelphia 5; 7 Grape St., Shaftesbury Ave., London, W.C. 2, England, 1952.

This book fills a long recognized need for an orthopedic text in medical schools and private libraries of physicians. The author is to be congratulated on the organization of his subject matter and the clarity and simplicity with which it is set forth, as well as the wise choice of illustrative and tabular material. He has condensed the material into 1,000 pages, using 463 figures and tables. The book is divided into four sections as follows: first, Introduction and Basic Principles of Orthopedic Practice; second, Regional Orthopedics; third, Orthopedic Disorders, and fourth, Neurology in Relation to Orthopedic Practice. The contributing authors are as follows: Dr. Fritz Cramer, who wrote the section on neurology related to orthopedics; Dr. Donovan J. McCune, who contributed notable chapters on many orthopedic subjects; Dr. Orren Babb, who discussed fractures and trauma to the elbow, forearm, and Dr. Littler, who contributed a fine chapter on the conditions of the hand and wrist.

The history of medicine and surgery is ably condensed in the first chapter. An eight-page reference at the end of this chapter will be of great value to all readers of the book, especially to those teaching orthopedics.

The author correlates the structural and the clinical anatomy, which is helpful to the student. He presents an excellent table on the ossification of the skeleton and a helpful kinesthetic chart for the joints. The illustrations of the structural anatomy, though small, are clearly marked, accurate, and particularly well chosen.

In the chapter on examination and diagnosis, he describes the method of eliciting common orthopedic complaints and relates the physical findings to the medical history and to the body as a whole. He illustrates joint range of motion and methods of muscle testing, together with common deformities to be observed. The routine laboratory procedures are discussed. He includes a table of roentgenographic views of orthopedic significance from the optional and routine standpoints.

In the chapter on treatment, the general principles of rest, support, posture in bed, general nutrition, drugs, preoperative and postoperative care, and ambulation of the surgical patient are discussed. All the common forms of traction are illustrated and clearly described in the text, together with brief indications for their use. A discussion of various types of casts and cast care is included. The common orthopedic operations are discussed from the standpoint of indication and technique. Important modifications are mentioned.

In the section on regional orthopedics, the chapter on posture and Dr. Littler's chapter on the hand and wrist are particularly well done. The chapters on the elbow, the shoulder, and the shoulder girdle are disproportionately brief and should have been expanded.

The section on orthopedic disorders is commendable in that the author describes the important as well as the rare conditions of the musculoskeletal system, the proper emphasis being placed on the more common and more important diseases. The problem of classification of the rare diseases is here evident, but that of the author is acceptable. The major peripheral vascular diseases are classified and discussed. The chapters on bone infections, metabolic disease, and neoplasms are particularly well done. Each important disease is given sufficient treatment to give the reader a concise picture of each entity. In the chapter on trauma, the author describes the more important fractures and discusses briefly their treatment.

Dr. Kramer's section on neurology concludes the text. In the 10 chapters of this section, he discusses neuroanatomy, physiology, the neurological examination, and the laboratory procedures together with various radiographic techniques of neurological significance. He then discusses diseases and injuries of the brain, cord, and peripheral nerves. The illustrations, diagrams, and tabular material are fundamental and excellent.

This reviewer commends this book to the profession for the basic knowledge and the fundamental treatment of diseases, injuries, and disorders of the musculoskeletal system.

The Kidney: Medical and Surgical Diseases. By Arthur C. Allen. Price, $15. Pp. 583. Grune & Stratton, Inc., 381 4th Ave., New York 16, 1951.

This comprehensive treatise combines in one volume a summary of our present clinical and pathological knowledge of the medical and surgical diseases of the kidney. There are also liberal references at the end of each section to original and experimental work. Because of the author's vast experience in the field of kidney pathology, most of the discussion naturally is built around his own observations and views. However, the views of others are given thorough consideration.

The format of the book is excellent. It is bound in water-repellent cloth, the paper is of high quality, the type is very clear, and there are extremely few typographical errors for a book of this size. It is profusely illustrated with literally hundreds of plates, including drawings and photographs of gross and microscopic specimens from the author's vast collection.

The book is divided into sections on embryology, anatomy, physiology, congenital malformations, clinical diseases of the various anatomical divisions of the kidneys, calculi, and tumors. Of greatest interest to practicing pediatricians are the sections on congenital malformations, acute and chronic nephritis, and renal rickets.

Since about 70% of all cases of acute diffuse glomerular nephritis begin in the period between 5 and 20 years of age, the author's discussion of this condition is of particular interest to pediatricians. There is a discussion of the pathogenetic and clinical aspects of the disease, as well as the pathology. He agrees that the bulk of our present evidence suggests that acute diffuse glomerulitis results from changes in the glomerular capillaries due to altered tissue reactivity, hypersensitivity, or some other form of antigen-immune body reactions.

Most present-day pathologists agree with Bell that the clinical entity known as lipoid nephrosis, or nephrotic syndrome, is actually a glomerulitis, and Dr. Allen is no exception. Pathologically, according to Allen, clinical lipoid nephrosis is actually chronic membranous or chronic lobular glomerulonephritis. Several pages of discussion and many fine plates are used to develop this concept. When lipoid nephrosis was first described it was often called "tubular nephritis," but Allen states that, although there are minor tubular changes, the essential findings are those of a glomerulitis.

In a large section on diseases of the tubules, there is a description of the many forms of "toxic" nephritis. Most of these are classified as proximal nephron nephrosis. He classifies "lower nephron nephrosis" as hemoglobinuric nephrosis involving proximal and distal nephrons.

Although this book is not one that the busy pediatric practitioner can use as a pocket reference, it is certainly a valuable reference text for anyone in any way interested in diseases of the kidney.

Childhood Problems and the Teacher. By Charlotte Buhler, Faith Smitter, and Sybil Richardson. Price, $3.75. Pp. 372, with 41 illustrations. Henry Holt & Company, Inc., 257 4th Ave., New York 10, 1952.

This book is a 1952 example of the collaboration of disciplines in an effort to understand children who are difficult to educate.

Charlotte Buhler, professor of psychiatry, Faith Smitter, director of child guidance, and Sybil Richardson, cooordinator of research, have analyzed the behavior problems of 114 children in a public school system.

Specific problems studied are distributed through ages 1 to 17 years, with nearly equal division of sexes. Problem behavior in children is interpreted from both biological and psychological aspects. Patterns of symptoms which occur together are presented to demonstrate what a teacher may handle successfully herself and what she should refer to outside authorities. It is pointed out that a teacher transmits to the children the culture of their time and of their country. Children who fail to react successfully to demands of our culture in the school system develop problem behavior. It is the teacher's responsibility to evaluate the nature and gravity of the problem. Some of the problems analyzed by the authors had to do with rejection of school, truancy, lack of interest, poor learning ability, cheating, aggressive behavior, withdrawing, and psychosomatic complaints. Forty-one drawings made by children are reproduced in the book as illustrations.

As stated in the preface, this book attempts to do the following three things: (1) to give the teacher an understanding of the dynamics of behavior problems that he may observe; (2) to determine by means of case examples what the teacher may achieve in various circumstances, and (3) to describe the type of problem with which the teacher needs specialized assistance.

An inventory of cases by problems as well as an index of subjects makes the book useful for reference. Although written for teachers who want to apply principles of modern psychology and psychiatry to solve the problems of children growing up in our complex western world, this book will provide valuable reading for parents, doctors, social workers, and others who deal with children.

Electrocardiographic Studies in Normal Infants and Children. By Robert F. Zeigler, M.D. Price, $10.50. Pp. 195, with 297 illustrations. Charles C Thomas, Publisher, 301-327 E. Lawrence Ave., Springfield, Ill., 1952.

This work represents a prodigious amount of painstaking minute observation. It represents a detailed study of electrocardiograms in infancy and early childhood. The author clears many controversial issues. This volume is a must for anyone reading the electrocardiograms of infants or small children.

Vaccination par le B. C. G. By R. Mande and A. Heut. Price, $7.50. Pp. 168, with 48 illustrations. Le Centre International de L'Enfance, Paris, France, 1952.

This book contains the answers to many questions that have arisen about tuberculosis immunization by vaccination with BCG. The method of selection of subjects for vaccination by means of the tests for tuberculosis is thoroughly explained, as well as the various methods of testing. The different techniques of vaccination are illustrated, with the study of postvaccinal allergy and the complications that may arise. These are all illustrated by excellent photographs. The principal complications seem to be cicatrix of the scar and various forms of adenitis occasionally resulting in suppuration.

The second part of the book deals with factors that may influence the vitality or activity of BCG and the results of its use in the different provinces of France.

This is a very well written and well organized book. Although it is in French, it is very easily read, and the illustrations are self explanatory. The subject matter is such that it should be read by all having an interest in this very important prophylaxis.

Advances in Pediatrics. Volume 5. Edited by S. Z. Levine, M.D. Price, $7. Pp. 273, with 58 illustrations. Year Book Publishers, Inc., 200 E. Illinois St., Chicago 11, 1952.

The fifth volume of "Advances in Pediatrics" includes six monographs. The subjects are varied, including Bacterial Meningitis, by Alexander, Vitamin K Deficiency in Hemorrhagic Diseases of the New Born, Angiocardiographic Studies in Children, Iron Metabolism in Infants and Children, BCG Vaccination, by Wallgren, and the Nephrotic Syndrome.

All the subjects are discussed in great detail and obviously from authoritative viewpoints. Bibliographies are extensive. The volume forms an up-to-date and complete refresher course in each of the subjects covered. No one except a research worker would need to know more about these topics than is presented here. The book should be read by all physicians who work with children.

Allergic Pruritus: Its Dermatologic Management. Edited by Stephan Epstein, M.D. Price, $2.50. Pp. 76 with 12 plates. The Bruce Publishing Company, 2642 University Ave., St. Paul 4, Minn., 1952.

This small volume is a collection of essays which were presented as a "Symposium on Itching Dermatoses," at the meeting of the American College of Allergists in St. Louis in 1950. Seven prominent dermatologists have contributed chapters, and, although these are well written and should be of value to dermatologists, the book does not contain much of especial interest to pediatricians, with the exception of Webster's chapter of 12 pages, which treats in detail of the various measures used to relieve itching, and Epstein's chapter of 13 pages on "bacterial eczema,"

which applies very directly to the treatment of eczema in infants and young children. Any pediatrician who is especially interested in eczema would do well to buy the book and study this chapter.

The Rhesus Factor. By G. Fulton Roberts, M.A., M.D. Price, 5 shillings. Pp. 90, with 7 tables. William Heinemann, Ltd., 99 Great Russell St., London, W.C. 1, 1952.

The third edition of this book has brought it quite up to date. The first three chapters are devoted to a description of the antigen, the antibody, and the diseases caused by the factor. The next chapter is a consideration of the treatment and is very complete. The remaining seven chapters deal with the incomplete antibody, nomenclature, the problems in hemolytic disease, the laboratory techniques, and the routine study of specimens.

While the book is intended as a brief and elementary survey of the subject, this edition has broadened the subject so that all that is necessary is enclosed in this compact little volume. It is very well written and greatly lessens the gap between the clinician and the laboratory worker.

The Other Child: The Brain-Injured Child. By Richard S. Lewis, Alfred A. Strauss, and Laura E. Lehtinen. Price, $2.50. Pp. 108, Grune & Stratton, Inc., 381 4th Ave., New York 16, 1951.

The impact that a child with organic brain disease has upon the family constellation may be more damaging to the family than to the child itself. An understanding by the parents of the true nature of the injury and its prognosis is necessary for proper adjustment of the family to their problem. This book, by Lewis, Strauss and Lehtinen, is an attempt to interpret to the parents the difference between their normal children and this "other child."

The book describes in nontechnical language and by use of many examples the nature of the thinking and learning processes in such a child and the necessity for the proper and realistic evaluation. The special problems of education, in which the authors are especially qualified, are well handled.

The book assumes that the reader has a certain amount of orientation in the field of psychology and is written for the intelligent parent. It is of value not only to parents, but also to teachers, social workers, psychologists, doctors, and any others who must cope with the problems of families with brain-injured children.

Growth and Development of Children. Edited by Ernest H. Watson, M.D. and George H. Lowrey, M.D. Price, $5.75. Pp. 260, with 41 tables and 54 figures. Year Book Publishers, Inc., 200 E. Illinois St., Chicago 11, 1951.

This little book fills a long-felt need for a concise work on this very basic and important subject. Apparently the successful teaching of growth and development has been one of the weak links in pediatric education.

Though this is a brief treatise on a broad subject, it should serve well the medical student, the intern, the resident, and even the busy practitioner.

The following chapters are covered in an interesting and adequate manner: Heredity and Environmental Factors in Growth and Development; Fetal Growth and Development; Normal Physical Measurements; Behavioral Development; Organ Development; Osseous Development; Role of the Endocrine Glands in Normal Growth and Development; Energy Metabolism; Nutrition in Normal Growth, and An Outline of Abnormal Growth.

The Physiology of the Newborn Infant. Edition 2. By Clement A. Smith, M.D. Price, $7.50. Pp. 335, with 44 figures. Charles C Thomas, Publisher, 301-327 E. Lawrence Ave., Springfield, Ill., 1951.

The original edition of Dr. Clement A. Smith's book, printed in 1945, satisfied the most stringent criteria of excellence. It has taken its place as one of the most scholarly, reliable, and delightfully readable scientific volumes of recent years. At the time of its introduction originality could be added to this listing of attributes, since no previous effort had been made to bring together what was known of the physiology of the fetus and the newborn. During six years this work has evolved as a standard reference. The format of the second edition, which is now

available, has not been changed, nor have new chapters been added; rather, each section has been carefully revised in the light of current knowledge. Clinical summaries and excellent complete bibliographies at the end of each chapter remain as particularly attractive features. Not only is an extraordinary amount of information presented, but also the data pertinent to a particular subject are critically evaluated, and their application to other related topics is pointed out. Whether one is interested in typical electrocardiographic variations in the newborn infant, the cyclic rhythmicity of respiration in the premature infant, the ability of the neonate to digest and metabolize fat, the increment of increased circulating blood volume attributable to delayed clamping of the umbilical cord, blood group incompatibilities, or the problems presented by infants born of diabetic mothers, to cite only a few examples, what is known can be found in "The Physiology of the Newborn Infant."

Untoward Reactions of Cortisone and ACTH. By Vincent J. Derbes, M.D., F.A.C.P. and Thomas E. Weiss, M.D. Price, $2.25. Pp. 69. Charles C Thomas, Publisher, 301-327 E. Lawrence Ave., Springfield, Ill., 1951.

By reviewing recent literature the authors present an account of untoward effects which may occur during therapy with cortisone or corticotropin (ACTH). The discussions of these reactions give little insight into their mechanisms, and several misleading statements occur. The action of cortisone which leads to osteoporosis and spontaneous fractures is not the same action which makes it useful in preventing fibrous tissue formation in traumatized joints as stated in the introduction. While the negative calcium and phosphorus balance during cortisone therapy is described, its physiological basis and, hence, its rational treatment are ignored. To speak of the "direct action of cortisone on the heart. . ." is hardly justified when describing the return of heart size to normal in Addison's disease. No details are included in the chapter on treatment. The use of stimulating doses of corticotropin periodically in patients on long-term cortisone therapy to prevent adrenal atrophy is not mentioned. The book cannot be recommended.

Annual Review of Medicine. By Windsor C. Cutting. Price, $6. Pp. 442. Annual Reviews, Inc., Stanford, Calif., 1952.

This annual volume presents an excellent review of the literature on 19 fields of medicine. The material is presented in readable fashion and is excellently annotated. In many instances the comments of the editors of the individual sections add a personal touch which is helpful in evaluating the material presented. Certain sections, such as the one on infectious diseases, have summaries which would make valuable additions to all sections. The chapter on laboratory aids to diagnosis and therapy is actually a discussion on hematology rather than a presentation on laboratory diagnosis.

As this volume is primarily a review of the literature presented in an impartial fashion, personal expressions of opinion are minimized. This is an excellent reference volume to bring the reader up to date on recent developments in the specific entities included in this book.

Speech Habilitation in Cerebral Palsy. By Marion T. Cass. Price, $3. Pp. 212. Columbia University Press, 2960 Broadway, New York 27, 1951.

The title is a good one, since the word "habilitation" indicates that we are dealing not with individuals who once had normal speech, but with those in whom speech was never normal. Except for its use in the title, however, the author continually refers to "rehabilitation" in the text.

The author obviously has had a great deal of experience in the field of teaching cerebral palsied children. However, the book would have been up-to-date 10 or 15 years ago but at the present moment is out-dated, particularly so far as the discussion of the medical aspects is concerned. Practically all references are to work which was done prior to 1943.

The first two chapters of the book deal with the medical background of brain injuries, and these represent a great deal of work and study on the part of the author. However, they are not very relevant to the subject at hand and are definitely out-dated. The concepts on neuro-

physiology are out-moded. There are some errors in statements made which may be attributed to typographical inaccuracies. The third chapter deals with the general motor reeducation of the cerebral palsied child, and also is out-dated and contains many controversial statements.

The best portion is the next three chapters, which deal with the problem of speech, with which the author is best acquainted. The author's concept that the child should be treated as a whole is an excellent one, and the manner of making an inventory of the child's abilities is also very good. The methodology offered for teaching relaxation (upon which the author places much emphasis), for controlled breathing, and for voluntary control of muscles involved in speech is excellent. However, some of the recommendations are too general. The author believes that the sequence in teaching should parallel speech development in the normal child, a thesis which is logical and has theoretical validity. However, the speech clinician will often find that it is best to start with what the child is able to do, rather than what he should be able to do first. The author also emphasizes the need for acoustic training in children, and her points are well made. She stresses that there are fundamental differences in training the various types of cerebral palsy and the methods must be adjusted to these differences; yet the speech training which she offers does not allow for these differences in diagnosis. It is obvious that the differences in therapy are not dependent upon the diagnostic category (e. g., spastic or athetoid), but rather upon the inventory of speech defects made by the therapist on the individual case.

There is a good chapter covering educational problems, with suggestions for a proper setup for educational programs, and an appendix which gives a form used by the author in speech testing of children with cerebral palsy.

The book as a whole is an excellent one for those interested in fundamental suggestions in the treatment of cerebral palsied children with speech difficulties. (It is, of course, true that what is said about the child with cerebral palsy is also true of other types of children with speech defects.) The book might be considered as too technical for one who is not actually going into speech therapy of cerebral palsy, but not informative enough for one who wishes to specialize in the speech treatment of children with cerebral palsy.

It is unfortunate that no illustrations have been added to this book, because there are areas where they would be rather illuminating. The book would have been improved by a better printing job, since it is rather difficult to read.

Directory of Pediatric Societies *

INTERNATIONAL

International Union for Child Welfare

President: Mrs. Gordon Morier.
Secretary: Dr. Georges Thélin, 16, rue du Mont-Blanc, Geneva, Switzerland.
Place: Bombay, India. Time: Dec. 5-12, 1952.

IV° Congreso Pan-Americano de Pediatria

President: Dr. Leonel Gonzaga.
General Secretary: Dr. Adamastor Barbosa.
Secretariat: Rua Dias da Rocha 25, ap. 602, Copacabana, Rio de Janeiro.
Place. Rio de Janeiro. Time: 1954.

IV° Congreso Sudamericano de Pediatria, Confederacion Sudamericano de Sociedades de Pediatria

President: Prof. Carlos Prado.
Vice President: Dr. Marcelo Garcia.
General Secretary: Dr. Alvaro Aguiar.
Place: Avenida Mem de Sá 197, Rio de Janeiro. Time: 1954.

FOREIGN

Academy of Medicine, Toronto, Section of Paediatrics

Chairman: Dr. Bernard Laski, Toronto.
Secretary: Dr. Crawford Anglin, Toronto.
Place: Academy of Medicine.

Australian Paediatric Association

President: Dr. Robert Southby.
Secretary: Dr. J. Colebatch, 12 Collins St., Melbourne.
Place: Canberra. Time: Yearly in April.

Sociedade Brasiliera de Pediatria

For officers, see IV° Congreso Sudamericano de Pediatria, above.

British Paediatric Association

President: Prof. J. Smellie.
Secretary: Prof. Alan Moncrieff, Institute of Child Health, The Hospital for Sick Children, Great Ormond St., W. C. 1, London.
Place: Windermere. Time: Between Easter and Whitsunday.

Canadian Paediatric Society

President: Dr. D. B. Leitch, 210 McLeod Bldg., Edmonton, Al.
Secretary: Dr. R. L. Denton, 1615 Cedar Ave., Montreal 25.

Danish Pediatric Society

President: Dr. A. Rothe-Meyer, Children's Hospital Fûglebakken, Copenhagen F.
Secretary: Dr. E. Winge Flensborg, Children's Hospital Mantinsvej, Copenhagen V.
Place: Different hospitals. Time: First Wednesday of every month.

*Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

Departmento de Pediatria da Associação Paulista de Medicina

President: Dr. Oswaldo Riedel de Souza e Silva.
Secretaries: Dr. Ovidio Collesi.
Dr. Helda Elina Vieira de Barros.
Place: Avenida Brigadeiro Luiz Antonio, 278, São Paulo, Brasil.

Deutsche Gesellschaft für Kinderheilkunde

President: Prof. Dr. Adam, Universitats Kinderklinik, Erlangen.
Secretary: Prof. Dr. Jochims, Städtische Kinderklinik, Lübeck.
Place: Bayreuth. **Time:** Sept. 1-4, 1952.

Hellenic Society of Pediatrics

President: Dr. Spiros Charocopos, Alexandras Ave. 1, Athens.
Secretary: Dr. Nikas Zervos.
Place: Amphitheater, Medical School of the University of Athens. **Time:** Once a month.

Nederlandse Vereniging voor Kindergeneeskunde

President: Dr. G. J. Huët, 39 Brediusweg, Bussum.
Secretary: Dr. H. Reerink, % Sophia Kinderziekenhuis, 160 Gordelweg, Rotterdam.
Place: Children's Hospital or Pediatric Clinic. **Time:** Three times a year.

Norsk Pediatrisk Selskap

President: Dr. Arne Njå, Sporveisgt. 29, Oslo.
Secretary: Dr. Helge Granrud, Abbedikollen 38, Bestun, Oslo.
Place: Barneklinikken, Rikshospitalet, Oslo. **Time:** Friday, six times a year.

Pediatric Society of New Zealand

President: Dr. Edward J. Cronin, Power Board Bldg., Queen St., Auckland.
Secretary: Dr. J. D. Matthews, 104 Symonds St., Auckland.
Place: Auckland. **Time:** Sept. 9, 1952.

Philippine Pediatric Society

President: Dr. Renato Ma. Guerrero.
Secretary: Dr. Felix A. Estrada.
Place: Santo Tomas University Hospital and other pediatric centers. **Time:** Every two months.

Royal Society of Medicine, Section of Paediatrics

President: Dr. D. W. Winnicott.
Secretary: Dr. J. J. Kempton; Dr. A. P. Norman.
Sectional Editor: Dr. D. MacCarthy
Place: 1 Wimpole St., London W.1. **Time:** Fourth Friday of each month, 4:15 p. m.

Schweizerische Gesellschaft für Pädiatrie

President: P. D. Dr. F. Bamatter, St.-Léger 8, Genève.
Secretary: Dr. M. Nicod, Place Bel-Air 2, Lausanne.

Sociedad Argentinade Pediatrie

President: Dr. Rudolfo Kreutzer.
Secretary: Dr. Luis M. Cucullu.
Place: Sante Fè 1178, Buenos Aires.

Sociedad Boliviano de Pediatria

President: Dr. Carlos Daza Meruvia.
Secretary: Dr. Cecilio Abela Deheza.
Place: S. C. I. S. P. Bldg. Time: Every Thursday.

Sociedad Chihuahuense de Pediatría

President: Dr. Alfonso Ruiz Escalona.
Secretary: Dr. Armando G. Herrera.
Place: Hospital Infantil del Estado, Chihuahua, Chih., México.

Sociedad Chilena de Pediatria

President: Prof. Dr. Raúl Gantes A.
Secretary General: Dr. Edmundo Cardemil.
Place: Medical Society, Merced 565, Santiago.

Sociedad Cubana De Pediatria

President: Prof. Agustin Castellanos y Gonzales.
Secretary: Dr. Carlos Hernandez Miyares, Apartado 2430, Habana.
Place: Catedra de Clinica Infantil Hospital Nuestra Senora de las Mercedes and Hospital Municipal de Infancia, Habana. Time: 9 p.m., last Wednesday of every month.

Sociedad de Pediatría de Concepción (Chile)

President: Dr. Próspero Arriagada.
Secretary: Dr. Guillermo Repetto D.
Place: Hospital Clinico Reginal Concepción. Time: 7:30 p.m., second Tuesday of every month, April to December, inclusive.

Sociedad de Pediatria del Litord

President: Dr. Raul Bruera.
Vice-President: Dr. Isidoro Slullitel.
Secretary General: Dr. Elsa Galimany.

Sociedad de Pediatria y Puericultura del Atlantico

President: Dr. Atilio Marino.
Vice-President: Dr. Helena Uribe de Jimenez.
Secretary-Teasurer: Dr. Hernando Borrios Angulo.

Sociedade Portugesa de Pediatria

President: Prof. Dr. Lucio de Almeida.
Vice-President: Dr. Cordeiro Ferreira.
Secretary General: Prof. Dr. Carlos Salazar de Sousa.
Treasurer: Dr. Mario Cordeiro.
Place: Clinica pediatrica do Hospital D. Estefania, Lisbon. Time: Monthly.

Sociedad Uruguaya de Pediatria

President: Dr. Euclides Peluffo.
Secretary: Dr. Ruben Gorlero Bacigalupi.
Place: Av. Agraciada 1464, piso 13, Montevideo.

SOCIEDAD VENEZOLANA DE PUERICULTURA Y PEDIATRÍA

President: Dr. Guillermo Rangel.
Secretary: Dr. Guillermo Tovar E., P.O. Box 5, Caracas.
Place: Hospital Municipal de Niños, Caracas. Time: Once a month.

SOCIEDAD YUCATECA DE PEDIATRIA

President: Dr. Humberto Perez Concha.
Secretary: Dr. Prudencio Ruiz Salazar.
Treasurer: Dr. Jorge Antiveros Andrade.

LA SOCIÉTÉ DE PÉDIATRIE DE MONTRÉAL

President: Dr. Henri Charbonneau, 3095 Sherbrooke, Montréal.
Secretary: Dr. Edmond Baril, 301 Carré St-Louis, Apt. 1, Montréal.
Time: Second Thursday of each month.

SOCIÉTÉ DE PÉDIATRIE DE PARIS

President: Prof. Jean Cathala, 45 rue Scheffer, Paris XVI.
Secretary: Prof. Maurice Lamy, 94 rue de Varenne, Paris VII.
Place: Hôpital des Enfants Malades, 149 rue de Sèvres, Paris XV. Time: 4 p.m., third Tuesday of each month.

SOCIÉTÉ DE PÉDIATRIE DE QUÉBEC

President: Dr. Roland Thibaudeau, 203 des Erables, Québec.
Secretary: Dr. Euclide Dechêne, 312 du Roi, Québec.
Place: Quebec. Time: May and November.

SWEDISH ASSOCIATION OF PEDIATRICS

President: Prof. Sture Siwe, Lund.
Secretary: Dr. Karl-Axel Melin, Färjestadsvägen 16, Bromma.

SWEDISH MEDICAL SOCIETY, SECTION FOR PEDIATRICS AND SCHOOL HYGIENE

President: Docent Urban Hjarne, Stockholm.
Secretary: Dr. M. d'Avignon, Pediatric Clinic, Karolinska Sjukhuset, Stockholm 60.
Place: Swedish Medical Society, Klara Östra Kyrkogata 10, Stockholm, and Pediatric Clinic, Upsala. Time: Second Friday of each month, except summer.

UNITED STATES

National

AMERICAN MEDICAL ASSOCIATION, SECTION ON PEDIATRICS

Chairman: Dr. Walter B. Stewart, 8 N. Tallahassee Ave., Atlantic City, N. J.
Secretary: Dr. Wyman C. C. Cole, 3001 W. Grand Blvd., Detroit.

AMERICAN ACADEMY OF PEDIATRICS

President: Dr. Warren W. Quillian, 140 Alhambra Circle, Coral Gables, Miami 34, Fla.
Secretary: Dr. E. H. Christopherson, 636 Church St., Evanston, Ill:
Place: Palmer House, Chicago. Time: Oct. 20-23, 1952.

SOCIETY FOR PEDIATRIC RESEARCH

President: Dr. Horace Hodes.
Secretary: Dr. Sydney S. Gellis.

The American Pediatric Society

President: Dr. Irvine McQuarrie, University of Minnesota Hospitals, Minneapolis.

Secretary: Dr. Aims C. McGuinness, 237 Medical Laboratories, University of Pennsylvania, Philadelphia 4.

Place: Hotel Traymore, Atlantic City, N. J.; May 6, 7, and 8, 1953.

Sectional

Intermountain Pediatric Society

President: Dr. John T. Mason.

Secretary: Dr. Stanley R. Child.

Place: Ambassador Athletic Club, Salt Lake City. Time: 7 p.m.

New England Pediatric Society

President: Dr. Albert W. Fellows.

Secretary: Dr. Harry Shwachman, 300 Longwood Ave., Boston.

Place: Boston, except Spring meeting. Time: Wednesdays, four times a year.

North Pacific Pediatric Society

President: Dr. Carl Ashley, 2455 N. W. Marshall St., Portland, Ore.

Secretary-Treasurer: Dr. S. Gorham Babson, 1107 S. W. 13th St., Portland, Ore.

Place: Sun Valley, Idaho (1952), Seattle (1953). Time: September, 1952, and March, 1953.

Northwestern Pediatric Society

President: Dr. F. G. Hedenstrom, 420 Lowry Medical Arts Bldg., St. Paul.

Secretary: Dr. Charles L. Steinberg, 1118 Lowry Medical Arts Bldg., St. Paul.

Place: Minneapolis; St. Paul; Duluth, Minn., and Rochester, Minn. Time: January, April, July, and October.

Rocky Mountain Pediatric Society

President: Dr. John Lichty, Pediatric Department, Colorado General Hospital, 4200 E. 9th Ave., Denver.

Secretary: Dr. Edwin T. Williams, 1850 Gilpin St., Denver 6.

Place: Announced by mail. Time: Monthly dinner meetings, October to June.

Southwestern Pediatric Society

President: Dr. Clement J. Molony, 416 N. Bedford Dr., Beverly Hills, Calif.

Secretary: Dr. Carl A. Erickson, 960 E. Green St., Pasadena, Calif.

Place: Jonathan Club of Los Angeles. Time: 6:30 p.m., second Wednesday in January, March, May, September, and November.

State

Alabama Pediatric Society

President: Dr. David B. Monsky, 748 Adams St., Montgomery 5.

Secretary: Dr. Alice H. Pye, 305 Church St., Montgomery.

Place: Montgomery.

Arizona Pediatric Society

President: Dr. Henry E. Running.

Secretary: Dr. William F. Schaffman.

Time: On Call.

California Medical Association, Section on Pediatrics

Chairman: Dr. Joseph W. St. Geme, 511 S. Bonnie Brae, Los Angeles 5.
Secretary: Dr. Alvin H. Jacobs, 2201 Webster St., San Francisco 15.
Assistant Secretary: Dr. Clement J. Molony, 416 N. Bedford Dr., Beverly Hills.

Florida State Pediatric Society

President: Dr. Charlotte C. Maguire, 1413 Kuhl Ave., Maguire Bldg., Orlando.
Secretary-Treasurer: Dr. Wesley S. Nock, 273 Alhambra Circle, Coral Gables.
Place: West Palm Beach. Time: Nov. 8 and 9, 1952.

Georgia Pediatric Society

President: Dr. John A. Simpson, Athens.
Secretary: Dr. J. Harry Lange, 490 Peachtree St., Atlanta.
Place: Savannah. Time: At time of annual state meeting.

Hezekiah Beardsley Pediatric Club of Connecticut

President: Dr. Joseph Hetzel, 51 Central Ave., Waterbury.
Secretary: Dr. James H. Root Jr., 103 N. Main St., Waterbury.
Time: With the Connecticut State Medical Society in the spring, with the Connecticut State Clinical Congress in the fall; winter meeting usually in December.

Illinois State Medical Society, Section on Pediatrics

Chairman: Howard R. Miller, 418 Jefferson Bldg., Peoria 2.
Secretary: Harry H. Boyle, 2376 E. 71st St., Chicago 49.

Indiana State Pediatric Society

President: Dr. Harold D. Lynch, Evansville.
Secretary: Dr. L. T. Meiks, Indianapolis.

Iowa Pediatric Society

President: Dr. John McKitterick, 315 Tama Bldg., Burlington.
Secretary: Dr. James Dunn, 501 Davenport Bank Bldg., Davenport.

Massachusetts Hospital Association

President: Dr. Philip D. Bonnet, Administrator, Massachusetts Memorial Hospitals, Boston.
Executive Secretary: Henry G. Brickman, 14 Somerset St., Boston 8.
Place: Boston. Time: Annual Meeting, January, 1953.

Michigan State Medical Society, Pediatric Section

President: Dr. Horace L. French, 301 Seymour Ave., Lansing.
Secretary: Dr. Park S. Bradshaw, 1014 Jefferson St., Muskegon.
Place: Detroit: Time: September, 1952.

Montana Pediatric Society

President: Dr. Orville M. Moore, 555 Fuller Ave., Helena.
Secretary: Dr. Joseph W. Brinkley, 410 Central Ave., Great Falls.
Place: Variable. Time: Spring and fall.

Nebraska Pediatric Society

President: Dr. Michael Crofoot.
Vice-President: Dr. Donald C. Nilsson.
Secretary-Treasurer: Dr. A. J. Lombardo, 401 Aquila Ct., Omaha.
Place: Children's Memorial Hospital. Time: 6 p.m., dinner, third Thursday of each month.

New Hampshire Pediatric Society

President: Dr. Douglass W. Walker, Laconia.
Secretary: Dr. Wm. D. Penhale, Concord.
Place. Alternately Concord, Hanover, Laconia, and Manchester. Time: Four times a year.

New Mexico Pediatrics Society

President: Dr. M. K. Wylder, Suite 14, Medical Arts Sq., Albuquerque.
Secretary: Dr. Ly Werner, Suite 25, Medical Arts Sq., Albuquerque.
Place: Variable. Time: Every two or three months.

North Carolina Pediatric Society

President: Dr. J. A. Shaw, 107 Bradford Ave., Fayettesville.
Secretary: Dr. Bailey Webb, 809 W. Chapel Hill St., Durham.
Place: Pinehurst. Time: May 6, 1952.

South Carolina Pediatric Society

President: Dr. Walter Moore Hart, 117 W. Cheves St., Florence.
Secretary: Dr. John R. Paul Jr., 82 Rutledge Ave., Charleston.
Place: Columbia, S. C. Time: Second Monday and Tuesday in September; Sept. 8 and 9, 1952.

Tennessee Pediatric Society

President: Dr. O. L. Von Canon, 846 McCallie Ave., Chattanooga.
Secretary: Dr. Oliver W. Hill, 4807 Newcom Ave., Knoxville.

Texas Pediatric Society

President: J. A. Bybee, Beaumont.
President-Elect: Dr. Robert L. Moore, Dallas.
Secretary: Dr. M. C. Carlisle, 1410 Austin Ave., Waco.
Treasurer: Dr. Jack M. Woodall, Big Springs.
Place: Dallas. Time: Oct. 17 and 18, 1952.

Virginia Pediatric Society

President: Dr. Edwin L. Kendig Jr.
Secretary: Dr. T. J. Humphries.

Wisconsin State Medical Society

Chairman: Dr. Franklin J. Mellencamp, 1513 E. Capitol Dr., Milwaukee 11.
Secretary: Dr. E. H. Pawsat, 44 S. Marr St., Fond du Lac.

Local

Academy of Medicine of Cleveland, Pediatric Section

Chairman: Dr. C. T. J. Dodge, 15457 Euclid Ave., Cleveland 12.
Secretary: Dr. Beulah Wells, 10515 Carnegie Ave., Cleveland 6.
Place: Allen Memorial Medical Library. Time: 8:15 p.m., October, December. February, and March.

Baltimore City Medical Society, Pediatric Section

President: Dr. Wilson Grubb, 4 E. 33d St., Baltimore 18.
Secretary: Dr. Gibson J. Wells, 3101 St. Paul St., Baltimore 18.
Place: 1211 Cathedral St., Baltimore 1. Time: 8:30 p.m., second Tuesday in November, January, and March.

Bronx Pediatric Society

President: Dr. M. H. Grand, 1749 Grand Concourse, New York.

Secretary: Dr. Charles T. Fried, 1220 Grand Concourse, New York.

Place: Morrisania City Hospital. Time: 8:30 p. m., second Wednesday of each month, except June, July, August, and September.

Brooklyn Academy of Pediatrics

President: Dr. Bernard Benjamin, 845 Park Pl., Brooklyn.

Secretary: Dr. Henry Cohen, 1761 E. 19th St., Brooklyn.

Place: Granada Hotel, Brooklyn. Time: 8:45 p.m., fourth Wednesday of February, March, April, October, and November.

Buffalo Pediatric Society

President: Dr. Douglas P. Arnold.

Secretary: Dr. Richard A. Downey.

Place: Children's Hospital, 219 Bryant St. Time: 8:30 p.m., first Monday of each month, September to June.

Central New York Pediatric Club

President: Dr. Frank Williams, 58 Swan St. S., Albany.

Secretary: Dr. Fred W. Bush, 325 Park Ave., Rochester.

Place: Various cities in New York State; fall meeting jointly with Upper New York State Academy of Pediatrics. Time: Third Tuesday of April and October.

Central Ohio Pediatric Society

President: Dr. E. V. Turner, 2479 E. Main St., Columbus.

Secretary: Dr. James Beasley, Lancaster.

Place: As announced. Time: Third Wednesday of September, November, January, March, and May.

Chicago Pediatric Society

President: Dr. George F. Munns.

Secretary: Dr. Matthew M. Steiner.

Place: Children's Memorial Hospital, 710 Fullerton Ave. Time: Third Tuesday of each month, October to May, inclusive.

Cincinnati Pediatric Society

President: Dr. Nicholas M. Kotona, 5771 Glenway Ave., Cincinnati 38.

Secretary: Dr. John E. Allen, 2546 Auburn Ave., Cincinnati 19.

Place: Children's Hospital, Elland Ave., Cincinnati. Time: On call.

Dade County Pediatric Society

President: Dr. Lynn Whelchel, DuPont Bldg., Miami, Fla.

Secretary: Dr. Howard Engle, 311 Lincoln Rd., Miami Beach, Fla.

Dallas Pediatric Society

President: Dr. Floyd A. Norman, 4325 Cole Ave., Dallas.

Secretary-Treasurer: Dr. Charles L. Smith, 4207 Lemmon Ave., Dallas.

Place: Children's Hospital of Texas. Time: 6:30 p.m., first Thursday in each month.

Detroit Pediatric Society

President: Dr. Ruben Meyer, 938 Maccabees Bldg., Detroit.
Secretary: Dr. W. W. Zuelzer, Children's Hospital of Michigan, Detroit.
Place: Variable. Time: Five times a year.

Honolulu County Pediatric Society

President: Dr. Teruo Yoshina, 1221 Victoria St., Honolulu.
Corresponding Secretary and Treasurer: Dr. John H. Peyton, Kauikeolani Children's Hospital, Honolulu.

Honolulu Pediatric Society

President: Dr. Teruo Yoshina, 1221 Victoria St., Honolulu.
Recording Secretary: Dr. M. H. Hasegawa.
Corresponding Secretary: Dr. John Peyton, Kauikeolani Children's Hospital, Honolulu.
Place: At doctors' homes in rotation. Time: 6 p.m., third Thursday of each month.

Houston Pediatric Society

President: Dr. Emanuel B. Brandes, 3101 Fannin St., Houston.
Secretary: Dr. Robert D. Lomas, 5101 Fannin St., Houston.
Place: River Oaks Country Club. Time: 7:30 p.m., fourth Tuesday of each month.

Kansas City Southwest Pediatric Society

President: Dr. Frank Hogue, Plaza Medical Bldg., Kansas City, Mo.
Secretary: Dr. S. J. Kranson, Truman Rd. and Ash, Independence, Mo.
Place: University Club, Kansas City, Mo. Time: On call.

Long Beach Pediatric Society

President: Dr. Kenneth Berkaw, 211 Cherry Ave., Long Beach, Calif.
Secretary: Dr. C. W. Latshaw, 3821 Atlantic Ave., Long Beach, Calif.
Place: Victor Hugo Restaurant, 730 E. Broadway. Time: First Tuesday night of every month.

Los Angeles County Medical Association, Pediatric Section

President: Dr. Sidney Rosin, 6230 Wilshire Blvd., Los Angeles.
Secretary: Dr. Arthur J. Holmes, 65 N. Madison Ave., Pasadena 1.
Place: Los Angeles County Medical Association Bldg., 1025 Wilshire Blvd., Los Angeles. Time: Second Thursday of alternate months beginning in September.

Medical Society of the County of Kings and Academy of Medicine of Brooklyn, Pediatric Section

President: Dr. Max Palinsky, 1270 45th St., Brooklyn 19.
Secretary: Dr. Bernard Greenberg, 670 Ocean Ave., Brooklyn 26.
Place: Kings County Medical Society Auditorium, 1313 Bedford Ave., Brooklyn. Time: 9 p.m., fourth Monday of January, March, April, October, and November.

Medical Society of the District of Columbia, Section on Pediatrics

President: Dr. George Maksim, 1418 Good Hope Rd. S. E., Washington 20.
Secretary: Dr. John E. Cassidy, 5402 Connecticut Ave. N.W., Washington.
Place: Medical Society Bldg., 1718 M St. N.W., Washington. Time: Spring and fall.

Memphis Pediatric Society

President: Dr. James G. Hughes, 20 S. Dunlap, Memphis.

Secretary: Dr. George S. Lovejoy, 609 N. Highland, Memphis.

Place: Rotated among four hospitals. **Time:** 7:30 p.m., second Tuesday of each month.

Milwaukee Pediatric Society

President: Dr. Franklin Mellencamp, 1513 E. Capitol Dr., Milwaukee.

Secretary: Dr. Karl Beck, 1411 Wauwatosa Ave., Milwaukee.

Place: Milwaukee University Club. **Time:** Second Wednesday of February, April, June, and October.

Mobile Pediatric Society

President: Dr. Daniel Sullivan, 1420 Government St., Mobile, Ala.

Secretary: Dr. Robert O. Harris III, 1514 Government St., Mobile, Ala.

Place: Admiral Semmes Hotel. **Time:** 7:30 p.m., fourth Thursday of September, November, January, March, and May.

Nashville Pediatric Society

President: Dr. Thomas Weaver, Bennie Dillon Bldg., Nashville, Tenn.

Secretary: Dr. Ethel Walker, 3415 West End Ave., Nashville, Tenn.

Place: Homes of members. **Time:** Third Thursday of every month.

Nassau Pediatric Society

President: Dr. Daniel Blitz, 1 Club Drive, Woodmere, N. Y.

Secretary-Treasurer: Dr. David J. Posner, 98 Wolcott Rd., Levittown, N. Y.

Place: Garden City Hotel, Garden City, L. I., N. Y. **Time:** 7:30 p. m., second Monday of October, December, February, April, and June.

New York Academy of Medicine, Section on Pediatrics

Chairman: Dr. Jerome L. Kohn, 50 W. 96th St., New York 25.

Secretary: Dr. Lawrence B. Slobody, 1 E. 105th St., New York 12.

Place: New York Academy of Medicine, 2 E. 103d St. **Time:** 8:30 p.m., second Thursday of each month, November to May, inclusive.

Northern California Pediatric Society

President: Dr. John A. Anderson, Stanford Hospital, 2351 Clay St., San Francisco 15.

Secretary: Dr. Edward Campion, 1018 E St., San Rafael.

Place: San Francisco. **Time:** Quarterly, as speakers are available.

Oklahoma City Pediatric Society

President: Dr. C. M. Bielstein, 301 N.W. 12th St., Oklahoma City.

Secretary: Dr. Louis Frank, 1200 N. Walker St., Oklahoma City.

Place: Oklahoma Club. **Time:** On call.

Philadelphia Pediatric Society

President: Dr. John B. Bartram, 2600 Lawrence St., Philadelphia 33.

Secretary: Dr. F. H. Harvie, 133 S. 36th St., Philadelphia 4.

Place: College of Physicians, 19 S. 22d St. **Time:** 8:30 p.m., second Tuesday from October to May, inclusive.

Pittsburgh Pediatric Society

President: Dr. Christian John Stoecklein, 519 Medical Arts Bldg., Pittsburgh 13.

Secretary-Treasurer: Dr. James W. Stirling, 694 Lincoln Ave., Pittsburgh 2.

Place: Western State Psychiatric Institute and Clinic. **Time:** Second Friday in October, December, February, and April.

Queens Pediatric Society

President: Dr. Samuel Dillon, 119-20 Rockaway Beach Blvd., Rockaway Park, N. Y.

Secretary: Dr. Moe Goldstein, 111-32 76th Ave., Forest Hills, N. Y.

Place: Seminole Club, 112th St. and 70th Rd., Forest Hills, N. Y. Time: Second Tuesday in October, December, February, and April.

Richmond Pediatric Society

President: Dr. Hobart T. Dougan, 1832 Monument Ave., Richmond 20, Va.

Secretary: Dr. Robert A. Hoffman, 1001 W. Franklin St., Richmond 20, Va.

Place: Medical College Hospital. Time: 8 p.m., third Thursday of each month, except June, July, and August.

San Antonio Pediatric Society

President: Dr. Edith M. Bonnet, 228 Medical Arts Bldg., San Antonio 5.

Secretary: Dr. Virginia S. Stovall, 1116 S. Texas Bldg., San Antonio 5.

Time: 7:30 p. m., supper, first Tuesday of each month.

Seattle Pediatric Society

President: Dr. O. William Anderson.

President-Elect: Dr. Robert Tidwell.

Secretary-Treasurer: Dr. James L. Tucker.

Place: College Club. Time: 6:30 p.m., fourth Friday of each month from September to June.

St. Louis Pediatric Society

President: Dr. Maurice J. Lonsway.

Secretary: Dr. John C. Herweg.

Place: Candlelight House, 7800 Clayton Rd. Time: 6:30 p.m., second Thursday of each month from October to May, inclusive.

University of Michigan Pediatric and Infectious Disease Society

President: Dr. Fred J. Chapin, 101 John St., Bay City.

Secretary-Treasurer: Dr. George H. Lowrey, University Hospital, Ann Arbor.

Place: University Hospital, Ann Arbor. Time: October, 1952.

Waco Pediatric Society

President: Dr. J. M. Garrett, 1408 Austin Ave., Waco, Texas.

Secretary: Dr. C. G. Shellenberger, 1318 Austin Ave., Waco, Texas.

Place: 1917 Austin Ave., Waco, Texas. Time: Third Tuesday of each month.

Westchester County Medical Society, Pediatric Section

President: Dr. Herbert Kahan, 650 Main St., New Rochelle, N. Y.

Secretary: Dr. Elvira Ostlund, 64 Highland Rd., Rye, N. Y.

Place: The Roger Smith Hotel, White Plains, N. Y. Time: Four dinner meetings a year, November, February, April, and June.

Western Michigan Pediatric Society

President: Dr. A. Morgan Hill, 310 Fulton, E., Grand Rapids.

Secretary: Dr. John C. Montgomery, 1810 Wealthy St., S.E., Grand Rapids.

Place: Two meetings in Grand Rapids, two elsewhere. Time: First Wednesday of October, November, April, and May.

Wichita Pediatric Society

President: Dr. Frank L. Menehan.

Secretary: Dr. R. C. Knappenberger.

Place: Homes of members. Time: 8:30 p.m., first Thursday, October through May.

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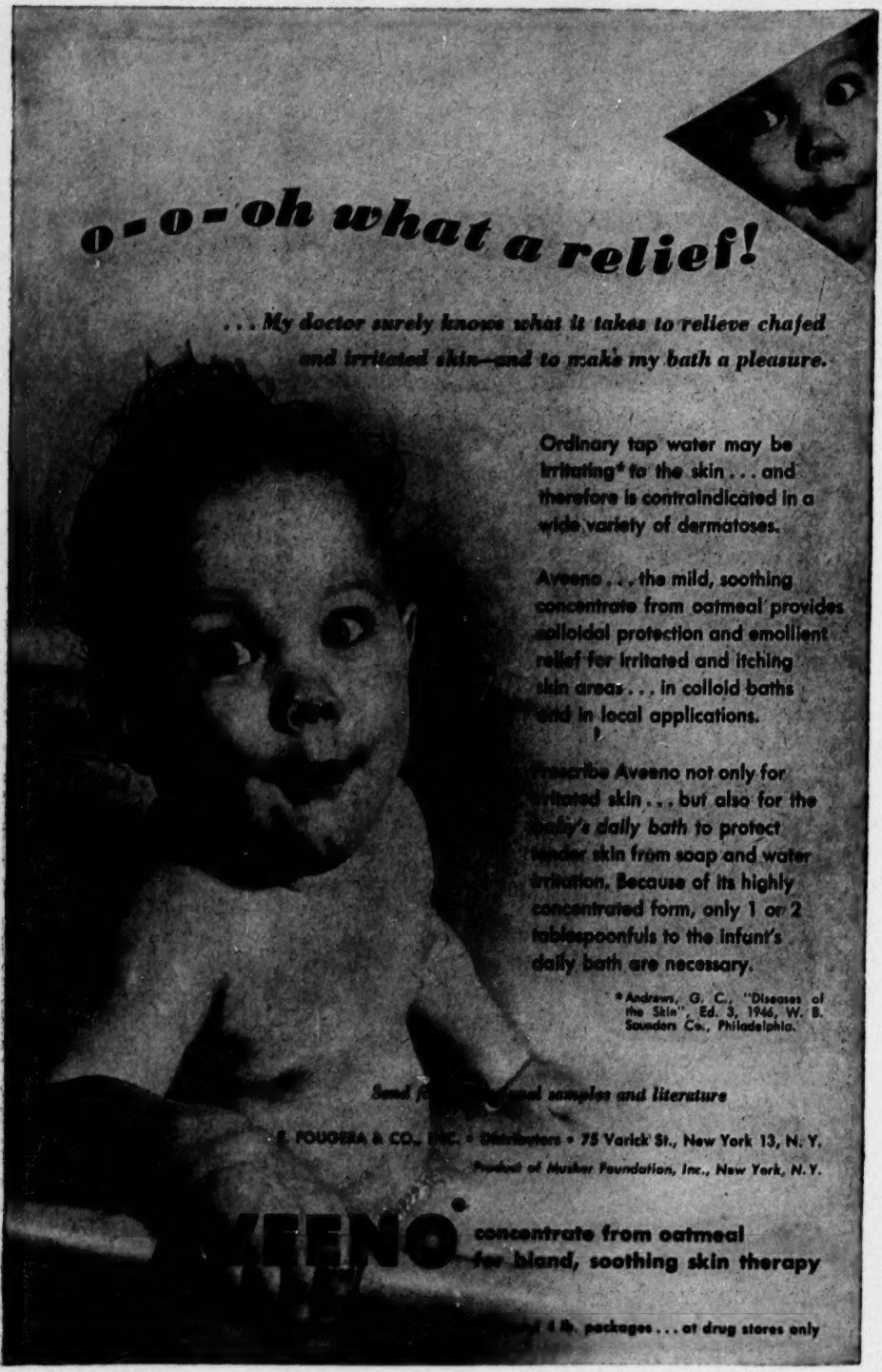
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* Andrews, G. C., "Diseases of the Skin", Ed. 3, 1946, W. B. Saunders Co., Philadelphia.
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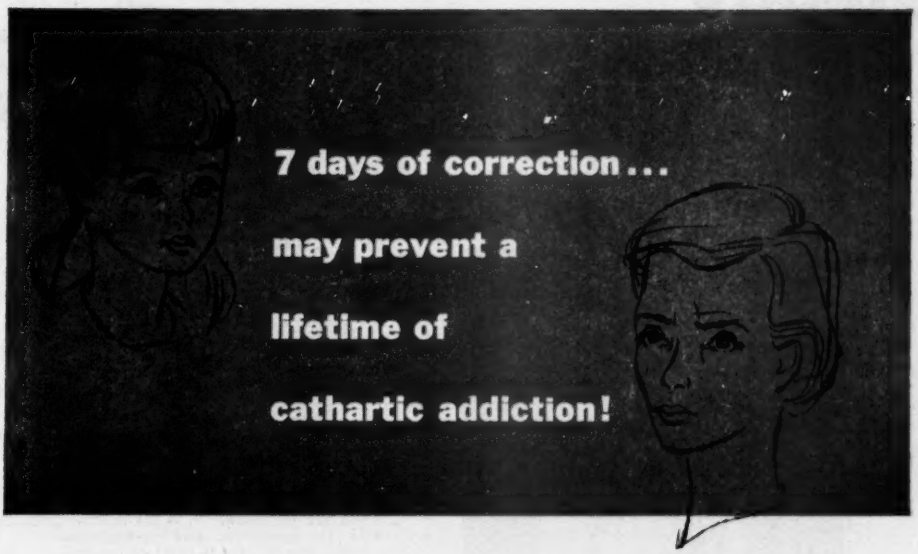

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1. Bockus, H. L.: Gastroenterology, Philadelphia, W. B. Saunders Co., 1943, vol. II, pp. 510-511.
2. Seidmon, E. E. P.: Am. J. Digest. Dis. *18*:274 (Sept.) 1951.
3. Mitchell-Nelson: Textbook of Pediatrics, ed. 5, Phila., W. B. Saunders Co., 1950, p. 808.
4. Schweig, K.: New York State J. Med. *48*: 1822 (Aug. 15) 1948.
5. Bargen, J. A.: Gastroenterology *13*:275 (Oct.) 1949.
6. Newey, J. A., and Goetzl, F. R.: Permanente Found. M. Bull. 7:2 (July) 1949.
7. Musick, V. H.: J. Oklahoma M. A. *43*:360 (Aug.) 1950.
8. Vignec, A. J., and Mitty, V. C.: J. Pediatrics *40*:576 (May) 1952.

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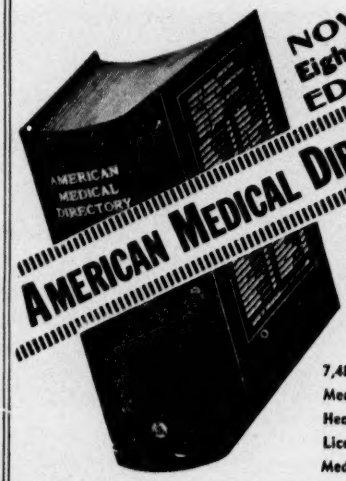

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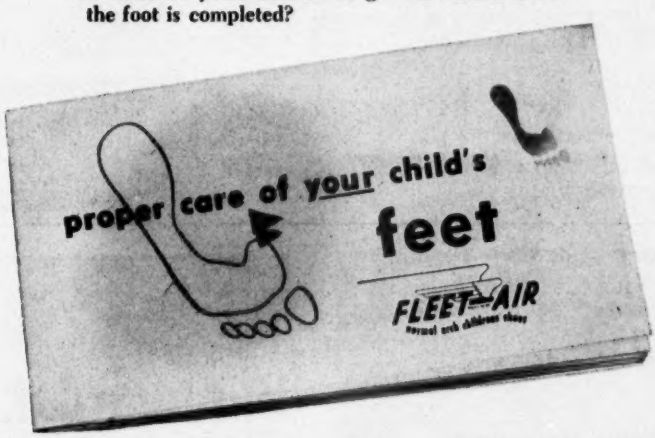

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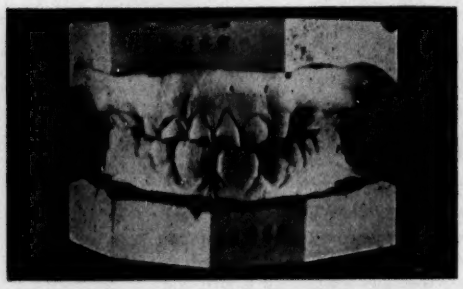

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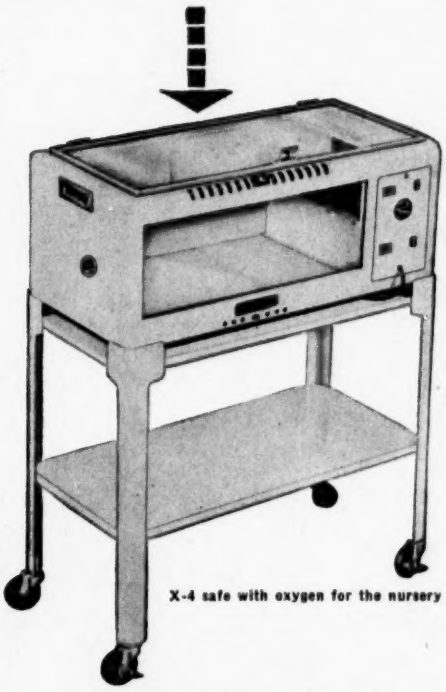

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